ON THE
ACTION AND SOUNDS
OF
THE HEART

GEORGE PATON, M.D.

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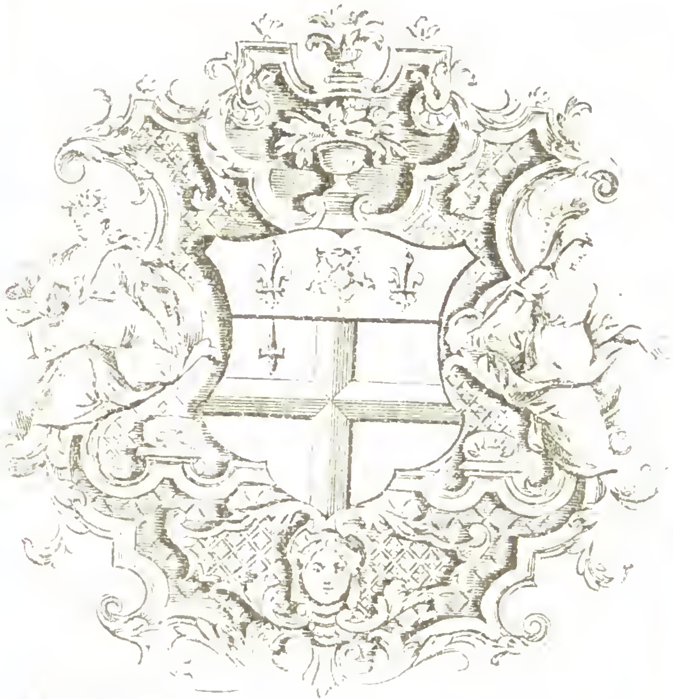

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RESEARCHES ON THE

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RESEARCHES

ON THE

ACTION AND SOUNDS OF THE HEART

By GEORGE PATON, M.D.

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PREFACE.

THE Author published a few years since a paper, in the *British Medical Journal*, on the Action of the Heart; and more recently, in the *Dublin Quarterly Journal of Medical Science*, another paper, on the Sounds of the Heart. These papers having been fully revised and enlarged, with much new matter added, are now published in the form of a distinct treatise—"Researches on the Action and Sounds of the Heart." The views advanced are based on a series of carefully conducted experiments on turtles, performed for several successive summers in Canada, during the highest temperature of the season, where every opportunity was afforded of listening to the sounds of the heart, and determining the manner in which they are produced.

London, Oct., 1872.

RESEARCHES

ON THE

ACTION AND SOUNDS OF THE HEART.

The opinion generally entertained by physiologists respecting the action of the heart is that the diastole of the ventricle is the result of its previous contraction; the fibres relaxing and assuming a state of rest. And the act is described as an instantaneous expansive movement, by which the ventricle returns to the same state with regard to form, size, and position, as during the previous period of repose; the blood flowing from the auricles by a suction-power, to fill up a vacuum produced in the interior of the ventricle.

If we examine the action of the heart when it is slow, pulsating at the rate of twelve or fourteen times a minute—in a turtle, for instance—we perceive that when the auricles contract and expel a portion of blood into the ventricle, it is immediately brought to the point of distention, and contracts, projecting a wave of blood along the aorta; or rather the contraction of the auricles extends by a sort of vermicular or continuous movement to the ventricle, causing it also to contract. After contraction the ventricle immediately dilates, its parietes becoming lax and flaccid, and blood passes rapidly from the dilated auricles, without contraction, into the cavity of the ventricle, filling it out to a certain extent.

Properly to understand the manner in which the diastole of the ventricle is effected during the action of the heart, we must remember that at this moment the veins in connection with the auricles, and the auricles themselves, are filled and almost distended with blood; and a *vis à tergo*, or power by which the blood is impelled towards the heart, is exerted against the internal parietes of the auricles, which, the instant the fibres of the ventricle and of the auriculo-ventricular valve begin to relax, causes the blood to enter the foramen of the ventricle, and quickly fill out or dilate its cavity to the previous extent during the pause. But when the action is more vigorous, the blood passes so quickly from the dilated auricles into the cavity of the ventricle, that the movements seem almost simultaneous, the blood appearing to open up, as it were, the relaxing parietes of the ventricle, or dilate them to their previous extent during the pause.

This constitutes the first stage of dilatation, the pause ensuing, during which the fibres of the auricles and of the ventricle remain relaxed. And as the auriculo-ventricular foramen continues open the *vis à tergo* exerted on the blood in the two cavities—the auricle and the ventricle—may be said to be nearly equalized. But if the force exerted by the blood as it passed from the auricle into the ventricle had been greater, it would have dilated the parietes till they reached the point of distention, and contraction would have ensued. But this power during the natural action of the heart is only produced by contraction of the auricles; for, as the blood on its return from the extremities passes along the venæ cavæ to the heart, it readily dilates and distends the parietes of the auricles, which being thinner, are more easily dilated than those of the ventricle; and then the auricle contracts, and projects a portion of blood into the ventricle, completing the second stage of the diastole, and causing the ventricles, as we have stated, to contract.

We have distinctly seen, during this state of the circulation, the blood as it reached the heart fill out and distend the auricles, causing them to contract and produce contraction in the ventricle, while no contractions were observed in the venæ cavæ, or extremities of the venous trunks. Showing that, when the action of the heart is slow, the impetus imparted by the blood as it reaches the auricles is sufficient to dilate the parietes, and produce contraction, independently of any action in the venæ cavæ, or extremities of the veins; and that one reason why the auricles are destitute of valves is, that the blood may readily reach and distend their parietes in maintaining the action of the heart.

There can be no doubt, however, that when the action of the heart is more vigorous the contraction of the extremities of the venous trunks exerts an influence over the distention of the auricles, whilst the valves with which the veins are furnished prevent the reflux of the blood during the auricular systole.

With regard to the amount of blood that enters the ventricle during each stage of the diastole, we may remark, that when the action of the heart is slow the ventricle is chiefly dilated during the first stage of the diastole, as the auricles do not contract with vigour, and in warm-blooded animals principally in the appendices; the act being apparently or chiefly designed to produce contraction in the ventricle, and consisting of a sort of vermicular movement, extending to the ventricle. When the action becomes more rapid, we have, during the second stage of the diastole, seen the ventricle enlarged by one-third of its previous size, the parietes being distinctly stretched out, and the fibres elongated, till distention was produced. From which it appears that in this condition of the circulation—that is, when the diastole consists of two stages—the greater proportion of blood enters the ventricle during the first stage of the diastole, and that the auricles only feebly and partially contract.

But when the action of the heart begins to increase, the auricles contract sooner in point of time and of rhythm. On being dilated they appear filled with blood, and a portion passes quickly from the auricles into the cavity of the ventricle, as its fibres relax after contraction. But, before the first stage of dilatation is completed the auricles contract, which annihilates the pause, and renders the diastole one continuous act. In this case we have distinctly seen and felt, in warm-blooded animals, the fibres of the ventricle, during the contraction of the auricles, continue to be extended and elongated till distention ensued, and contraction commenced.

When the action of the heart is still more vigorous, respiration being fully maintained, the contraction of the auricles is synchronous with the diastole of the ventricles. The auricles on being dilated, do not remain distended with blood, but immediately contract, and the whole of the blood that enters the ventricle is projected by contraction of the auricles. The *vis à tergo* in the veins has now no influence in assisting the dilatation of the ventricle, and the ventricle dilates whilst the auricles contract, and they contract with considerable energy, the diastole of the ventricle being completed by a continuous and uninterrupted movement, without the slightest appearance of a pause.

It is surprising to observe the different opinions that are entertained by physiologists on the action of the heart, according to the phenomena presented to their view. Dr. Hope states:* "On examining the posterior surface of the heart of a frog, when its action was reduced to 15 or 20 per minute, the whole of the auricle, which had previously been concealed by the ventricle, being now exposed, it was found that for a short space of time, the ventricle being at rest partially distended with blood, the auricle then contracted with a smart brief motion, but only partially contracted; for the sinus venosus was constantly full of blood, both in this experiment and those of the rabbit, and whether the circulation was quick or slow. When the auricle had relaxed again, and not till then, the ventricle, stimulated, I conjecture, by the motion, for it certainly was not by distention, was seen suddenly to rise up on its basis, to shorten its fibres, and to expel its contents, which latter action was slowly performed. After the completion of the systole, which was indicated by the pale colour, the diastole took place, and allowed a partial influx of blood, denoted by the return of the red colour; and in this state the ventricle remained quiescent for a short space, until again stimulated by the auricular contraction. It may be objected to this account, that as the action of the heart was preternaturally slow, the motions were abnormal. We thought, however, that we could discern the same series of actions, when the pulsations were 40 per minute." Professor Müller, as the result of his experiments on the action of the heart, states: "In the frog the contractions of the venous trunk, of the auricle, the ventricle and the bulbous aorta appeared to me to follow the order in which I have named the parts, the intervals between the four contractions being nearly equal; so that the same interval of time elapsed from the contraction of the auricles to the contraction of the ventricle, as between the contraction of the ventricle and that of the bulb of the aorta."

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* Hope on the "Heart," p. 11.

The discrepancy of opinion that exists between these two authors respecting the action of the heart, undoubtedly arose from the circumstance that Dr. Hope viewed the heart when its action was slow and languid; and Professor Müller when it was quick and vigorous, beating at the rate of 60 or 70 times per minute. And it will be observed that in the latter case the auricles contracted and expelled their blood into the ventricle whilst it dilated. And the movements being alternate, dilated whilst the ventricle contracted; so that nothing like a pause or period of repose occurred during the diastole of the ventricle. And this fact we have repeatedly witnessed in the course of our experiments in the frog, the fish, and the turtle, when the action of the heart was vigorous and respiration well maintained.

As the principle that obtains in the action of the heart, is the same in warm as in cold-blooded animals, Dr. Hope maintained that a pause always exists between the diastole of the ventricles and the contraction of the auricles, and that this contraction immediately precedes the contraction of the ventricles, or rather that it passes into it by a sort of vermicular movement. But other physiologists who have examined the action of the heart when it was vigorous in living animals, pulsating at the rate or 60 per minute and upwards, respiration being well maintained, are decidedly of opinion, that the contraction of the auricles takes place simultaneously with the commencement of the diastole of the ventricle; that no pause or period of repose occurs during the ventricular diastole, but that it consists of one distinct and continuous movement, immediately succeeded by the systole.

More fully to explain our views, we may observe that having denuded the heart of a living turtle, we carefully observed the phenomena. Respiration remained unaffected. When the heart beat at 10 or 12 pulsations per minute, the ventricle, after contraction, dilated, and became partially filled with blood passing into it from the distended auricles, a pause ensued; and then the auricles contracted and completed the diastole of the ventricle, causing it to contract. When the action increased to 16 or 18 pulsations per minute, the ventricle after contraction dilated, and a portion of blood passed from the distended auricles into the ventricle, filling out its parietes, but before this was completed, the auricles contracted, and projecting their blood into the ventricle brought it into a state of distention at once, which annihilated the pause, and rendered the diastole one continuous act. But on the pulsations increasing to 26, 28, and 30 per minute, the auricles contracted synchronously with the diastole of the ventricle, which consisted of one continuous and uninterrupted movement. The blood projected by the auricles filled out or dilated the ventricle, as its walls expanded, till distention ensued and contraction took place. Here we observed that, as the diastole proceeded, a sort of undulation passed along the cavity of the ventricle, as if the blood, by the force which it exerted, opened up and distended its walls. The animal remaining in a calm and quiescent state, and the action of the heart proceeding with much uniformity, suddenly as if impelled by an impulse, the auricles began to contract with great energy, and projected the blood into the ventricle with increased velocity; and its parietes expanded at a proportionate ratio, and contracted in a similar manner. The vigorous contraction of the auricles appeared to open up the relaxing parietes of the ventricle, and caused them to dilate with great velocity. These movements would continue for a time, and then the slower action of the heart would return.

In another turtle, the action of whose heart we examined during the highest temperature of the season (July 8th), it appeared that the contraction of the auricles exerted an influence on the dilatation of the ventricle; for, when the action of the heart had attained its greatest degree of speed—36 pulsations per minute—the contracting power of the ventricle was sometimes apparently prolonged as if from the vigour with which it acted; and whilst the ventricle continued in this contracted state, the auricles contracted, and the moment the blood entered the foramen of the ventricle, its parietes opened up, and continued to expand till they contracted. This fact was also apparent when the action of the heart was 30 pulsations per minute. We placed our fingers against the parietes of the ventricle, and we distinctly recognised that they did not become smooth and soft till the auricles began to contract; and the moment that took place, the blood appeared to open up the contracted walls of the ventricle, or caused them to expand till contraction commenced.

There was now a great difference in the character of the movements, both of the auricles and the ventricle. When the action of the heart was slow, as at 10 or 12 pulsations per minute, the auricles, on being dilated, continued distended with blood during the first stage of the diastole and the pause. But, when the action of the heart was vigorous, the auricles were dilating whilst the ventricle was contracting; and as the auricles immediately contracted on being dilated, the blood entered the foramen of the ventricle at the moment its fibres were beginning to relax, and rapidly dilated its walls, or filled out the cavity of the ventricle, till distention was produced, and contraction commenced.

We denuded the heart of a living frog, and carefully observed its action. The pulsations were 64 per minute. Respiration remained perfect. The auricles, on being dilated, immediately contracted, and they contracted synchronously with the diastole of the ventricle; the parietes of the ventricle continuing to expand as the blood was projected by the auricles, till distention was produced, and the ventricle contracted. During dilatation the ventricle assumed a deep red colour, and the blood could be distinctly seen to enter its cavity as the parietes expanded and dilated. During contraction the ventricle became small and pale, all the blood appearing to be expelled from its cavity. But, after contraction, there was no expansion of the parietes of the ventricle, and no blood entered its cavity till the auricles contracted. The animal continued to respire with vigour, and the action of the heart increased to 72 and 74 per minute. The movements were maintained in a similar manner, but the auricles contracted with greater rapidity; and as they projected the blood into the ventricle, its parietes expanded at a proportionate rate, and also more quickly contracted, projecting the blood with greater velocity along the aorta. After a few hours, the action of the heart fell to 30 per minute, and precisely the same movements were observed, the wave of auricular contraction passing as it were into the foramen of the ventricle; and the blood could be distinctly seen to enter its cavity, whilst the walls of the ventricle expanded and dilated. But there was not the slightest approach to anything like the dilatation of the ventricle before the contraction of the auricles.

On denuding the heart in warm-blooded animals we have carefully observed its action, both by touching the ventricles with the fingers and steadily watching their movements with the eye, the pulsations being in some cases, as in large animals, 64 per minute; in other cases, as in small animals, 80, 90, or 100 per minute. But at this rate of speed we have never been able to detect anything like a pause during the diastole of the ventricles. In all these cases the contraction of the auricles was synchronous with the diastole of the ventricles, which consisted of one rapid and continuous act, the blood being projected by the auricles into the cavities of the ventricles, till distention was produced and the systole took place, propelling the blood along the aorta and pulmonary artery.

We have now reached that degree of velocity in the action of the heart, when the auricles contract as the ventricles dilate; there being no interval between the diastole of the ventricles and the contraction of the auricles, but the acts occur simultaneously. And if we analyse these movements of the heart,—that is, when its action is quick,—we perceive that the ventricles do not relax before the auricles begin to contract, which can easily be ascertained by placing the point of the finger against the side of the ventricles, and observing the movements of the auricles. The contractile energy of the ventricles may even afford somewhat the appearance of a pause, the ventricle appearing to remain for an instant contracted, after it has propelled the wave of blood into the aorta, and we have even repeatedly seen, as already stated, the auricles contract, whilst the ventricle was still in a contracted state, but that moment the fibres relaxed, and as the blood from the auricles entered the ventricle, the parietes swelled out, and the diastole was completed in its usual manner by a continuous and uninterrupted movement.

When the action of the heart has attained such a degree of celerity that the contraction of the auricles is synchronous with the diastole of the ventricles, the parietes of the ventricles do not instantaneously expand, and the blood then enters the cavities. The movements are simultaneous. The diastole of the ventricles proceeds at a ratio proportionate to the energy with which the auricles contract. If the auricles contract with greater vigour, the ventricles dilate with greater rapidity. If the auricles contract with less vigour, the ventricles dilate with less. And if we carefully examine the parietes of the ventricles, we can distinctly see and feel the fibres gradually dilate as the blood from the auricles shoots to the apex and distends the cavity. No doubt the parietes of the ventricles relax after contraction in virtue of the power which they possess, as muscular tissue, of assuming their previous condition. But the fibres may not always relax with a degree of rapidity sufficient to sustain the vigorous action of the heart. Muscular fibres have been observed continuing in contraction.* And we have repeatedly seen when the animal became much agitated the ventricle remain contracted till excited to dilatation by the blood projected by the auricles. And Müller remarks that "the degree to which the heart dilates, independent of being dilated by a fluid, can be but slight." * We have also observed the auricles contract whilst the ventricle remained in a contracted state, the animal continuing calm and quiescent. And Laennec, in his observations on the action of the heart, states, "that in some cases the systole of the ventricles seems scarcely over before it begins again to swell out afresh."† The contraction of the auricles then, being synchronous with the diastole of the ventricles, subserves a most important purpose in maintaining the vigorous action of the heart. Then a force is exerted which converts the movement of relaxation into one of active dilatation, because the power with which the auricles contract may not only excite the dilatation, but impart a stimulus to the relaxing parietes of the ventricles, which renders the diastole the first and most prominent movement, and the systole its sequence. If we examine the action of the heart under these circumstances, we easily perceive that the movements of the ventricles partake of this character, that the ventricles first dilate and then contract. For the moment the auricles contract the ventricles swell out, as if by an active power; opening up or dilating their walls till fully distended, they contract, propelling the blood along the aorta and pulmonary artery. Fully to understand this movement, let us suppose a cavity into which a fluid flows as its walls relax or dilate, and when filled the cavity contracts and expels the fluid along another tube. The fluid enters the cavity as the walls expand, and is expelled as they contract,

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* Müller's "Physiology," vol. ii. p. 888.

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* Müller's "Physiology," vol. i. p. 233.

† Laennec on "Diseases of the Chest," p. 508.

which is the case when the action of the heart is slow. But suppose that a force is exerted when the fluid enters, opening up the dilating walls of the cavity, and when filled or distended the cavity contracts, expelling the fluid along another tube. It is evident that the whole character of the movement is changed. There is an active force exerted, both in dilating the cavity and contracting it to expel the fluid, and this is undoubtedly what obtains during the vigorous action of the heart.

That this is the principle on which the action of the heart depends, will more distinctly appear if we attend to the following points:—1st. The diastole of the ventricle consists of one continuous movement, by which the fibres are stretched out not merely to their normal extent, but till distention ensues and contraction commences. 2nd. The ventricle dilates with various degrees of rapidity, according to the velocity with which the circulation is maintained. It dilates with twice as great velocity at 60 per minute as at 30, and with three times as great velocity at 90 per minute, and so on in relative proportion to the action of the heart. And what is it that confers this power upon the ventricle—first of attaining during the act of dilatation to the point of distention and contraction; and secondly, of increasing the rapidity with which the fibres dilate, according to the increased vigour of the circulation? It cannot depend on mere muscular relaxation, for that would not carry it to the point of contraction; and the increased velocity of the dilatation of the ventricle cannot be accounted for on the principle of the fibres of the ventricle relaxing with greater rapidity according to the previous contraction, because increased action in the dilatation commences before the ventricle contracts. If the action of the heart be proceeding at a moderate rate of speed, and the auricles commence to contract with greater vigour, the ventricle at this moment dilates with greater velocity, and contracts with similar energy. Every successive movement, as the action of the heart increases, proceeds in the same manner, the first impulse of increased activity of the ventricle commencing with and depending on the rapidity of the diastole.

From these statements, then, it appears that the auricles during the vigorous action of the heart contract with energy, and exert an influence over the whole of the diastole of the ventricle. Many physiologists also concur in the opinion that the auricles contract with vigour. Dr. John Reid states "that the systole of the auricles is performed with great regularity when the action of the heart is still vigorous, and appears to be effected by the simultaneous contraction of all its fibres. According to the observations of Harvey, Lower, Senac, Haller, and others, the contraction of the auricles is performed with considerable force."* "In the experiments of Dr. Hope, Mr. Carlisle, Mr. Bouilland, and the Dublin Committee for Investigating the Cause of the Sounds of the Heart, the contraction of the auricles appeared to be comparatively trifling, and was most apparent in the appendices. From my own experiments," says he, "upon rabbits and dogs, I am convinced that the auricles contract considerably more when the movements of the heart are proceeding in a natural manner than some of these last experimenters would lead us to believe, and that this contraction is not confined to the appendix, but extends over the whole auricle; but that none of the muscular fibres of the auricles are passive, but exert a force proportionate to their strength, we have evidence both from experiment and the effect of disease. In some of those cases where an impediment to the passage of the blood from the auricle to the ventricle exists, all the muscular fibres of the auricles become much increased in thickness and in strength."

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* Article "Heart," by Dr. John Reid, in "Cyclopædia of Anatomy and Physiology." London.

There can be no doubt that during the moderate and regular action of the heart the auricles contract with energy. And we have the most satisfactory evidence for concluding that they exert a direct influence over the ventricle, both during its diastole and its systole, which enables us to see how the action of the heart is really increased and maintained. The physiologists who adopt the views supported by Dr. Hope, admit that the auricles are the primum mobile, or cause of the movements of the ventricle. But they confine their action to bringing the ventricle, already partially filled with blood, to the point of distention and producing the contraction; the first stage of dilatation and the pause according to that view always existing. So that as only an instant is occupied in bringing the ventricle to its full distention, it is chiefly in the rapidity of the contraction that the increased action of the heart is produced; which is a circuitous mode of increasing the circulation of the blood, and incompatible with the great velocity that is frequently attained. But in the preceding experiments we have shown that when the action of the heart is vigorous. the contraction of the auricles is synchronous with the diastole of the ventricle, which annihilates the first stage of dilatation and the pause. And as the force which the auricles exert extends through the whole period of the ventricular diastole, it increases the rapidity with which the parietes dilate, whilst it imparts to them a greater stimulus to contraction.

In this state of the circulation the ventricle is not only filled and distended solely by the contraction of the auricles, but it is brought to the point of distention by a smaller quantity of blood entering it than when the action of the heart is slow and languid. We have always observed that when the action of the heart is vigorous and the blood highly arterialized, the ventricle is more quickly distended and by a smaller amount of fluid; the vigorous condition of the ventricle rendering it susceptible of the finer stimulus. Thus we see how the action of the heart is increased and maintained when the systole of the auricle is synchronous with the diastole of the ventricle, the two movements proceeding in a regular and uninterrupted manner and in quick and rapid succession, and not only is this the case, but, when the auricles contract with still greater rapidity, the diastole of the ventricle is performed with greater energy, and the systole with similar velocity. Hence when the circulation is quick and vigorous the whole diastole of the ventricles, as well as the systole, is regulated by the auricles or directly under their influence, causing the ventricles to manifest an actively dilating power by the rapidity with which the movement is executed.

We perceive then that the principle on which the action of the heart is established, renders it adapted either to a slow, or a rapid circulation of the blood. When the action is slow a pause occurs in the diastole of the ventricle, which disappears as the circulation increases; and the auricular systole becomes synchronous with the ventricular diastole. If the ventricle pulsate at the rate of 14 or 16 times a minute, the pause occurs and is long, the blood being imperfectly arterialized; a due stimulus is not imparted to the heart, and all the functions of the body are languid. When the action of the heart increases to 30, 34, or 40 pulsations per minute in cold-blooded animals, as the turtle, and to 50 and 60 pulsations per minute in the warm-blooded, respiration being well maintained, the pause entirely disappears, and the diastole of the ventricle is synchronous with the contraction of the auricle, and occurs in half a second or thereabouts, and the systole in half a second. When the action increases to 90 pulsations per minute the diastole occupies one-third of a second or thereabouts, and the systole one-third, and the movements proceed in this manner, the diastole being quickly performed and succeeded by the systole; and when the action of the heart increases to 180 pulsations per minute, as in the menoculus pulex and some others, whose blood is highly arterialized, three pulsations occur in one second of time, allowing one-sixth for the diastole, and one-sixth for the systole.

But the ventricle is considered to dilate with a force which, in warm-blooded animals, is sufficient to open up the closed hand seizing it, a phenomenon that physiologists have had great difficulty in accounting for, and to which we shall particularly refer in another section.

We shall now shortly inquire how the diastole of the auricles is produced. The auricles, it is well known, are never empty of blood, a portion remaining after contraction, dilating their walls. The parietes of the auricles are thin, and during relaxation very distensible or easily dilated. The auricles possess no valves behind them, but a free communication exists between them and the extremities of the veins which are possessed of the power of contracting and dilating with the fibres of the heart. In cold-blooded animals, when the action of the heart is vigorous, the venous trunks or extremities of the veins contract first, then the auricles, after that the ventricle, and the bulb of the aorta, which is synchronous with the ventricular systole; the ventricle being still contracted when the aorta pulsates. In this case the contraction of the venous trunks distend the auricles with blood, and produce their contraction. But when the circulation was slower, we have observed the blood in the veins on its return from the body continue to distend the auricles and cause them to contract, independent of contraction in the venous trunks; showing that the blood returns along the veins with a power sufficient to distend the auricles and sustain the action of the heart, and this appeared to take place without a *vis-à-fronte;* for, in cases where respiration had ceased, and the ventricles were dilated and filled with blood, the blood on its return from the extremities continued to dilate the auricles and over-distend their parietes.

During these experiments the action of the heart was always increased by the animals continuing to respire for a time. The blood becoming more highly arterialized, appeared to impart a greater stimulus to the internal parietes of the auricles and ventricles. And it is a well-known fact that exercise or increased exertion quickens the circulation in the veins, and causes the blood to return with greater vigour to the heart.

But it appears that independently of this principle, the auricles are capable of being excited by a stimulus conveyed through the cerebral or sympathetic nerves to the heart. We have frequently seen in the turtle and also in other animals whose heart had been denuded, when the animal was remaining calm and quiescent, the action of the heart proceeding with great regularity, that suddenly, as if by a strong impulse, the auricles commenced to contract with great energy, and transmit the blood rapidly into the ventricle, increasing its action; and these movements would continue for a time, and then subside, the calm and uniform action of the heart being restored.

During our experiments on large warm-blooded animals, we have found the heart, immediately after being denuded, beat at 60 pulsations per minute and upwards. And on carefully examining its action, both by placing our fingers against its parietes, and observing its movements with the eye, we perceived that the blood passed rapidly along the veins to the auricles; and that the auricle on its parietes becoming tense communicated a strong impulse to the fingers that pressed it at the moment. Cruveilhier believes that it is during the dilatation of the auricles, that an expanding force is exerted by the parietes, but it is when distention has been attained, and the systole has commenced; for the fibres being shortened, the parietes are rendered tense; and by the force of the contraction expand, and are thrown outwards, communicating an impulse to the fingers.

We therefore conclude that the diastole of the auricles depends on the same principle, and is produced in a similar manner as the diastole of the ventricles; but that independent of this the auricles are susceptible of being excited by a stimulus conveyed through the nervous system, causing them to contract with greater vigour, and impart a stimulus to the ventricle, increasing the action of the heart.

CONTRACTION OF THE VENTRICLE.

When the ventricle commences to contract, its parietes are rapidly brought into the same plane with those of the aorta, so that the blood passes as it were along the same tube; and the contraction takes place with an impulse, the walls of the ventricle becoming tense and firm, and expanding with a force that communicates a shock to the fingers; and in large warm-blooded animals opens up the hand that seizes the ventricle, whilst the blood is being expelled from the cavity. During the diastole, the fibres of the ventricle are soft, smooth, and compressible: during the systole, tense, firm, and resisting. And if we examine this phenomenon in cold-blooded animals, where it can be most distinctly discerned, we perceive that the ventricle is first brought to the point of distention by the force of the blood from the auricles, and that when distended its parietes are still smooth and soft—beginning to grow tense. And as the contraction commences an impulse proceeds from the apex to the base, the fibres becoming rapidly firm and tense over all the ventricle, and the lateral parietes expanding with considerable force; the pressure of the fingers being always more resisted there than at the apex. And in large warm-blooded animals, when the action of the denuded heart was slow, we have observed, that as the auricles contracted, and sent their wave of blood into the ventricle, an undulation passed along the parietes, which still felt soft and compressible; but the moment the systole commenced, the parietes became suddenly tense and firm, opening up the hand that grasped them. And the force exerted by contraction of the auricles was not so strong as that of the lateral expansion of the ventricular parietes.

Physiologists, however, have connected this phenomenon with the diastole, rather than with the systole of the ventricles; believing that as the fingers are forced open, the ventricle must be dilating, and that it is inconsistent to suppose that the parietes could expand when the ventricle had commenced to contract and expel the blood from its cavity.

Hence Pechlin, Perrault, Bichat, Cruveilhier, and several other physiologists who have observed the action of the heart when it was exposed in living animals, considered that it is during the diastole of the ventricle, when it is receiving an accession of blood into the cavity, that the hand is opened up.

Dr. J. B. Williams in his observations on the action of the heart, states, that the diastole of the ventricle in large warm-blooded animals, in whatever manner it may be accounted for, takes place with a force sufficient to open up the closed hand. And Dr. Hope observes, "that at each systole, the sudden tension of the ventricle was such as to produce an abrupt shock to the fingers placed on any part of it, with which shock the first sound exactly coincided; and the impulse from lateral expansion was greatest at the margins of the auricular orifices, there throwing out the fingers with a sudden jerk." And the Dublin committee in the course of their investigations into the action of the heart, remark, "The ventricles with a rapid motion assumed a somewhat globular form in their middle part, and during their continuance in this state, they were hard to the touch, and if grasped by the hand at the commencement of the movement they communicated a shock or impulse, and separated the fingers."

In these observations, Pechlin, Perrault, Bichat, Cruveilhier, and Dr. Williams, speak of the ventricle as opening up the hand during the diastole. Whilst Dr. Hope, in describing the same act, speaks of it as occurring during the systole of the ventricle. The shock imparted to the hand coincided exactly with the first sound, and we know that it occurs during the contraction of the ventricle. And the Dublin committee state that the ventricle was hard to the touch, that is, in a state of tension, and consequently contracting when the shock or impulse was imparted to the hand.

These statements show the great difficulty that physiologists have had in classifying this movement, whether to refer it to the diastole or the systole of the ventricle, which undoubtedly arises from the manner in which the action takes place—the ventricle feeling to the touch as if it were dilating, whilst it is contracting with an impulse.

In explanation of this fact we may observe, that the longitudinal axis of the ventricle is shortened during contraction, and the parietes of the ventricle, though strongly muscular, partake much during life of the character of fibrous elastic tissue, expanding by the force of the contractile energy exerted. And when the ventricle is brought to the point of distention by the blood from the auricles, another force is immediately gained as the contractile energy commences. Just as an artery always reacts with greater force than that by which it is excited, so the ventricle, whilst a constant ratio exists between its power and that of the auricles, contracts with greater energy than it receives from them; and the left ventricle, being more muscular, contracts with greater force than the right. Hence, when the ventricle gives the impulse that communicates the shock to the hand, the parietes being then rendered tense, are thrown outwards and expand, from the force of the contractile energy exerted; the strong contractile action that shortens the ventricle, producing the lateral expansion, whilst the blood is propelled into the aorta.

We denuded the heart of a turtle, June 3rd, and on the action amounting to 32 pulsations per minute, the auricles contracting synchronously with the commencement of the diastole of the ventricle, we observed, that as the auricle projected the blood into the ventricle, it swelled out and dilated till it attained the point of distention ; it then immediately contracted, its parietes expanding with considerable force, resisting the pressure of the fingers applied to them. This was more particularly the case at the base of the ventricle and with the transverse diameter than the longitudinal axis of the ventricle. The longitudinal axis was distinctly shortened as the transverse diameter was increased. But at this moment every portion of the parietes felt tense; and this condition increased as the blood was expelled, the ventricle being contracted towards the orifice of the aorta. In another turtle where the circulation had ceased, but the auricles and ventricle continued for a time to contract and dilate with considerable energy, on applying a stimulus to the ventricle whilst it was soft and relaxed, and exciting the systole, the parietes quickly became tense, and expanded with a force that separated the fingers and thumb, as the ventricle contracted. And then the parietes suddenly relaxed, and felt soft and flaccid. On being again excited by the stimulus or by the irritation produced by contraction of the auricles, the movements were repeated, the parietes of the ventricle becoming tense and firm, and expanding in a similar manner: which phenomenon could not be produced by the impetus of the blood against the internal parietes of the ventricle, for only a small portion was now contained in the ventricle. But the parietes were here distinctly contracting as they expanded and threw out the fingers. And in our experiments on large warm-blooded animals, when the action of the denuded heart was maintained with great vigour, beating at the rate of 64 and 66 pulsations per minute, on grasping the ventricle with the hand, we perceived that on the auricles contracting, and sending the blood with an impetus into the ventricle, the parietes were rapidly brought to the point of distention, and as the contractile action commenced the parietes expanded with a force that opened up the hand, the walls being rendered more apart, whilst the ventricle was slightly curved in the centre, and the apex tilted up. And when the ventricle has been exposed in large warm-blooded animals, as the horse, and the circulation is brought to a stand, it is found that the ventricle continues susceptible of a stimulus for a short time, and on being irritated starts up at intervals, and its walls expand with a force that opens up the hand grasping them; which act is viewed by physiologists as the dilatation of the ventricle, but great difficulty has been experienced in accounting for the force with which it is performed. It cannot depend on the relaxation of the muscular fibres, for it is a general law that the parietes are then soft and compressible. And it cannot be produced by the impetus of the blood from the auricles, for the circulation has now ceased through both cavities. Besides, this phenomenon takes place totally independent of the auricles, on the stimulus or irritation being applied to the parietes of the ventricle itself. Hence many physiologists have thought that the ventricle is possessed of an actively dilating power inherent in the parietes by which this force is exerted. Whilst others have supposed that there are two sets of fibres connected with the ventricle—one for contraction, and another for dilatation. But the phenomenon admits of an easy explanation—the ventricle is now contracting and not dilating.

When the ventricle remains at rest, it is in a dilated state, its fibres soft, lax, and compressible. But when it starts up on being irritated, it is in a contracting state, its parietes becoming firm and tense, opening up the hand as its transverse diameter expands on its longitudinal axis being shortened. If the circulation had been maintained the blood would have passed into the ventricle from the auricle, when its walls are lax and flaccid, and on being brought to the point of distention, contraction would have commenced and been accomplished in the manner in which it is now performed. But when we see the ventricle empty of blood on being irritated suddenly become tense and expand with force, resisting the pressure of the fingers, we imagine it is dilating, but it is actually contracting at the moment.

There are two things then that particularly demand our attention. 1. The ventricle at this moment passes from a dilated or relaxed state into one that is firm and tense; and it is not the impetus of the blood from the auricles against the internal parietes of the ventricle that gives the shock or impetus to the hand, but it is on the parietes becoming tense, that is, by the fibres contracting, that the shock is communicated and the fingers thrown out. 2. The ventricle is a hollow muscle distensible, and in some measure possessed of the power of elastic tissue, and does not bring its sides and parietes into close apposition in expelling the blood from the cavity, but contracts from its apicial extremity to its base, and as its longitudinal fibres are shortened, its lateral parietes, becoming tense, are thrown outward and expand from the force exerted, and as this takes place with an impulse, the blood is rapidly propelled into the aorta, and the aorta instantly pulsates. It is, then, in the shortening of the ventricle whilst the parietes are simultaneously rendered tense, that the phenomenon of lateral expansion is produced, by which the shock is communicated to the hand. And the rapid shortening of the fibres depends on the strong contractile impulse exerted, in the propulsion of the blood-wave from the ventricle into the aorta.

SOUNDS OF THE HEART.

In every class of animals in which we have examined the action of the heart, we have found that the ventricle contracts towards the orifice of the aorta or vessel that conveys the blood from the ventricle into the system; the mode of contraction being evidently designed to facilitate the passage of the blood from the respective ventricle into the aorta or pulmonary artery.

In contracting, the ventricle does not continue its action till the whole of the blood is expelled. It only contracts to an extent proportionate with the impetus received from the auricles, expelling a certain amount of blood termed the blood-wave.

June 29.—Took a large turtle, and removed a portion of the sternum or bone that covers the thorax, leaving the thoracic parietes uninjured; and observed the action and listened to the sounds of the heart.

When the ventricle contracts and propels the blood into the aorta, it raises up slightly the parietes of the thorax, and we can distinctly follow the course of the blood from the ventricle into the arch of the aorta, by the heaving up or elevation of the muscular parietes. There appears to be two impulses or movements, first, that of the contraction of the ventricle; second, that of the distention and reaction of the aorta, which take place simultaneously, and are synchronous with the first sound of the heart.

The heart having been fully denuded, the animal continued strong and vigorous, moving about with great energy, and survived several days. Pulsations, 32 per minute.

We applied the stethoscope and listened to the sounds of the heart. Both sounds are distinctly heard; the first—a dull and prolonged sound terminating by a sort of knock, or as if a piece of cloth or leather were fully stretched out, or extended to its ultimatum—is heard most distinctly over the semilunar valves, at the origin of the aorta or a little above it, and synchronous with the contraction of the ventricle and pulsation of the aorta. The second sound immediately follows the first; is a short, sharp sound, like that produced by the tongue striking the roof of the mouth, and occurs during the contraction of the auricles, as they pour their blood with force into the ventricles during the diastole of the latter.

When the ventricle contracts, the blood passes rapidly into the aorta, which becomes more curved and tense, the contraction proceeding from the apex to the base, and as it reaches the muscular fibres around the origin of the aorta they contract with energy, passing below the semilunar valves, constricting the part, and sending the last portion of the blood-wave into the aorta with force; the aorta starts up, and the rapid reaction of the distended parietes causes the blood to recoil against the semilunar valves and shut them, as it imparts an impulse to the wave, the fibres of the ventricle being contracted behind the aorta, as if supporting the valves, whilst the blood recoils against them—synchronous with which the first sound of the heart attains intensity and terminates.

The sound commences as the blood passes with force through the aortic foramen, and terminates in the aorta, as the blood is thrown back against the valves and shuts them, whilst the aorta pulsates. Sometimes a little roughness or bruit attends this sound, which disappears as the ventricle contracts with greater vigour. The sound is distinctly heard through the medium of the stethoscope, or by the naked ear applied over the heart, and as many as 30 pulsations can be counted at a time without interruption.

Immediately after the contraction of the ventricle and impulse of the aorta terminate, the auricles contract; and as they propel their blood into the ventricle, the second sound of the heart is heard. It is a short, sharp sound, distinctly recognised through the medium of the stethoscope. It appears to be deeper-seated, and not so near the ear as the first sound, and is heard at the side of the aorta, in the position of the auricles pouring their blood into the ventricle. The whole of the auricles now contract, and not a part of them, and as they pour their blood with force into the ventricle, the second sound of the heart is produced.

July 3rd.—Denuded the heart of a large turtle; observed the action, and listened to the sounds.

When the ventricle contracts, it sends with an impulse the wave of blood into the aorta, distending its walls, and the aorta simultaneously contracts from its origin, shutting the valves and rendering the arch more curved as the blood is impelled along it, the ventricle being contracted when the aorta pulsates. Synchronous with the contraction of the ventricle and pulsation of the aorta, the first sound is heard, and is produced by the blood being propelled with force through the aortic foramen and the wave that has entered the aorta recoiling against the valves as it is impelled onwards. Hence the sound is dull and prolonged, and heard most distinctly over the semilunar valves as they shut. Immediately after the aorta pulsates, the auricles contract with an impulse, and as they propel the blood into the ventricle through the ventricular foramen, the second sound is heard. It is a short, sharp sound, and appears at a greater distance than the first sound, as the auricles are here deeper seated than the arch of the distended aorta.

The first sound ascends in the course of the blood from the ventricle along the aorta. But the second sound descends in the path of the blood from the auricles into the ventricle; so that it is heard in a lower position than the first sound.

The second sound is recognised at the commencement of the contraction of the auricles, immediately following the first sound, and is most distinct over the auriculo-ventricular foramen, as the blood is sent with force into the ventricle distending its *walls*. And a longer interval of silence occurs between the second, and the commencement of the first sound; because the ventricle on being distended, commences to contract with an impulse towards the aorta, and the first sound is produced as the blood is propelled with force through the aortic foramen and the aorta pulsates at its origin, shutting the valves.

On the vigorous action of the heart, the auricles which have been refilling during contraction of the ventricle commence to contract immediately after the aorta pulsates and the first sound terminates. But when the action of the heart grows slower the auricles are later in commencing to contract, so that they appear rather to precede immediately the succeeding contraction of the ventricle than succeed the preceding ventricular contraction. And it is this circumstance of viewing the action of the heart when it is becoming slower, that has led experimentalists to believe that the contraction of the auricles could not account for the second sound of the heart, which so immediately follows the first sound.

We tested this question of the sounds of the heart with the greatest care and attention, when the pericardium had just been slit open, and the action of the heart strong and vigorous. Pulsations 40 per minute. Temperature of day 92° Fahrenheit, in the shade. On applying the stethoscope and carefully avoiding pressure, we counted 1, 2, 3, 4, 5, 6, &c., as we heard the first sound of the heart, and several gentlemen who observed the action, distinctly perceived that the ventricle contracted and the origin of the aorta pulsated as we mentioned the numbers. And on listening to the second sound, it was also clearly found that the auricles contracted as we counted 1, 2, 3, 4, 5, 6, &c., thus putting it beyond all doubt that the first sound of the heart is produced during contraction of the ventricle and pulsation of the origin of the aorta; and the second sound is produced during contraction of the auricles, as the blood is propelled into the ventricle.

The ventricle then contracts, propelling the blood-wave with force into the aorta, distending its walls, and the aorta simultaneously reacts from its origin, shutting the valves as it gives the impulse to the wave. And the first sound of the heart is distinctly heard over the semilunar valves, whilst the ventricle is still contracted and the arch of the aorta more curved and tense. And the second sound is heard immediately after the first as the auricles contract and send the blood with an impulse into the ventricle.

We performed similar experiments on several other turtles during the highest temperature of the season, and with precisely the same results. In some cases the action of the heart amounted to 36 pulsations per minute, in others to 40 and even 50 per minute; but in general 36 to 40 pulsations per minute were the highest attained.

The ventricle in contracting propelled its wave of blood with force into the aorta, distending its walls, and the aorta, reacting with a power superior to that by which it was distended,* caused the blood to recoil against the semilunar valves and shut them, as it imparted its impetus to the wave, the ventricle being contracted behind the valves, when the impulse of the aorta is exerted, synchronous with which the first sound of the heart is distinctly recognised over the semilunar valves, or a little above that; the impulse of the aorta preceding the pulse at the extremity of the arteries by a very short interval.

The fibres of the ventricle relax as the auricles contract, and the parietes of the aorta at the same time become straighter, and attain an interval of rest, till the ventricle contracts and again distends the aorta, there being the closest connection between the condition of the ventricle and that of the aorta.

Dr. Hope, as the result of his experiments on the sounds of the heart, concluded that the first sound depends on three causes: First, a degree of valvular sound, produced by the blood recoiling against the auriculo-ventricular valves; second, a sudden jerking extension of the muscular walls of the ventricle, as a sound is produced by the extension of the leather of a pair of bellows, which he termed the sound of extension; third, a prolongation or increase of this sound by the sonorous vibrations peculiar to muscular fibre. Dr. Williams maintains that the first sound of the heart is produced chiefly by the muscular contraction itself, and that as a general law a sound accompanies every rapid muscular contraction; but he considers that the tightening of the auriculo-ventricular valves also contributes to the production of the first sound.

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* Poisseuille.

The Dublin Committee, as the result of their experiments on the sounds of the heart, concluded that the first sound is produced either by the rapid passage of the blood along the internal surface of the ventricles on its way to the mouths of the arteries, or by the *bruit musculaire* itself, or probably by both these causes combined; whilst the London Committee attributed the first sound to muscular contraction, but considered that the impulse of the heart against the thorax may occasionally act as an auxiliary cause in increasing the intensity of the sound. M. Cruveilhier, as the result of his observations, states that the first sound is heard most distinctly at the origin of the large arteries, and diminishes as we approach the apex of the heart. And Carpenter concludes that the principal cause of the first sound exists at the entrances to the arterial trunks. And it does not seem, he states, that any other reason can be assigned for it than the prolonged rush of blood through their orifices, and the throwing back of the semilunar valves; which, in suddenly flapping down again, produce the second sound.*

From these statements it appears that physiologists have, in general, confined the first sound of the heart to the action of the ventricle; but that M. Cruveilhier and Carpenter believe that it is connected with the entrance of the blood into the aorta and pulmonary artery—a view which we consider the nearest approach to the real cause of the first sound of the heart.

We must further observe, it is considered an established fact in physiology, that the first sound of the heart is synchronous with the contraction of the ventricle, and also with the pulsation of the aorta, or as it is termed, with the pulse of the arteries near the heart. Carpenter says * that "the first sound is evidently synchronous with the impulse of the heart against the parietes of the chest, and also with the pulse as felt near the heart." Dr. Wood states, † "The first sound is heard during the contraction or systole of the ventricle, and is synchronous with the beating of the ventricle, and with the pulsation in the large arteries near the centre of circulation, but anticipates by a very minute, but still appreciable interval, the pulse of the wrist." Müller observes,‡ "The pulsation of the arteries near the heart is synchronous with the systole of the ventricle." And Dr. Hope maintains § that, "synchronous with the systole of the ventricle are, the first sound, the impulse of the apex against the ribs, and in vessels near the heart, the pulse; but in vessels at some distance as the radial, the pulse follows at a barely appreciable interval." From this united testimony of physiologists, it must be admitted that the first sound of the heart is synchronous with the contraction of the ventricle and pulsation of the aorta.

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* Carpenter's "Physiology," p. 419.

But the aorta cannot pulsate without the blood recoiling against the semilunar valves, and shutting them as it is impelled onwards. If the blood had been sent along the aorta in a constant stream, there would have been no shutting of the valves in its onward course. But the blood is propelled by the ventricle,

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* Carpenter's "Physiology," p. 418.

† Wood's "Medicine." Philadelphia.

‡ Müller's "Physiology," p. 699.

§ Hope on "The Heart," p. 56.

par saccadés, in successive waves, and as each wave is sent with force into the aorta, it distends its walls, and the aorta instantly reacts, shutting the valves by the rebounding of the wave as it is impelled onwards. And the ventricle is fully contracted at the origin of the aorta, as if supporting the valves, whilst this impulsive movement takes place. The distending force of the ventricle ceases as the last portion of the wave enters the aorta, but it is now within the valves, and the parietes return on their contents with an energy superior to that by which they were distended, throwing the blood against the valves and shutting them, as an impulse is exerted on the wave; the impulse of the aorta being succeeded by the pulse at the wrist by only an appreciable interval of about one-thirtieth of a second.* Hence, it is during the onward course of the blood that it recoils against the aortic valves, just as the fluid in the forcing pump reacts against the valves, during the lateral pressure that propels it forward.

We have seen in the course of our experiments that the first sound of the heart is heard most distinctly over the semilunar valves, at the origin of the aorta, and occurs as the aorta pulsates and the valves shut. During the ventricular systole the blood is sent rapidly into the aorta, rendering it more curved and tense; and as the contraction reaches the muscular fibres at the origin of the aorta they contract with energy, passing behind the semilunar valves, constricting the part, whilst the aorta becomes fully distended and reacts, throwing the blood against the valves, and shutting them, by the impetus given to the wave.* Immediately after this, the ventricle commences to dilate as the auricles contract, and the parietes of the aorta grow straighter, and recover their former situation, till the ventricle again contracts, and the movements are repeated.

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* Müller's "Physiology," p. 176.

These facts seem to establish the proposition that the blood recoils against the aortic valves and shuts them during the ventricular systole, and not during its diastole; and that the sound which is then produced is the first sound of the heart. It commences as the blood is propelled with force through the aortic foramen, and attains its intensity in the aorta, as it reacts on the advancing wave. It is a dull and prolonged sound, fully brought out at its termination; synchronous with the contraction of the ventricle and impulse of the aorta, and precedes the pulse at the extremities of the arteries by only an appreciable instant.

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* The fact that during the ventricular systole, the blood recoils against the aortic valves and shuts them, on pulsation of the aorta can easily be verified, by denuding the heart of a living fish, or other cold-blooded animal, in summer, and observing the phenomena.

May 16th.—Denuded the heart of a perch fish, and carefully observed its action.

When the ventricle contracted, it started up and assumed a curved form, propelling the blood-wave into the bulbous aorta or vessel that conveys the blood from the ventricle to the gills—distending its walls: the ventricle falling a little backward, as the bulbous aorta swelled out and pulsated, the blood-wave being then distinctly seen to pass along the arch. The impulse of this aorta took place as quick as the eye could follow it from contraction of the ventricle, the ventricle being still contracted, whilst the aorta pulsated, and in shutting the valves the aorta raised itself from its base, becoming smaller and narrower as it propelled the blood onward. Immediately after this, the parietes of the ventricle relaxed as the auricle contracted, and the walls of the aorta became soft, till again distended by the blood-wave from the ventricle. But there was no shutting of the valves, and no recoiling of the blood against them, except when the bulbous aorta pulsated, and that was synchronous with the contraction of the ventricle.

And we can perceive the necessity of the semilunar valves being shut, and sustained by the ventricle contracted behind them, as the full impetus of the distended aorta is imparted to the blood-wave,—the impulse recoiling against the valves, and passing along the tube with such rapidity that the amount of blood which enters the aorta almost simultaneously displaces a similar amount at the extremities of the arteries,—communicated to the veins.

In contraction, the force which the ventricle exerts is spent on the impetus or momentum given to the wave and the distention of the aortic parietes; but as an artery always reacts with increased vigour, the latter force is more than regained and exerted during the arterial systole.

No portion of the first sound could be connected during these experiments with the auriculo-ventricular valves, because a membranous expansion, very small, of the internal parietes, or lining membrane of the ventricle is extended over the orifices, and covers the parts completely during the contraction and expansion of the walls of the ventricle; and it is so small, and so situated, that no sound can be produced at the part during the ventricular contraction.

The beat of the facial artery, says Müller, which is known to depend on the contraction of the ventricle, is nearly synchronous with the heart's impulse, being only one-thirtieth of a second later than it, while the second sound is not heard until one-fifth of a second after the impulse; consequently the second sound can in no respect be connected with the pulsation of the aorta, where, as we have shown, a sound is produced. "The second sound," according to Dr. Hope, "results from the sudden expansion of the semilunar valves, occasioned by the reflux on them of the columns of blood in the aorta and pulmonary artery during the ventricular diastole." And Dr. Williams says: "The second sound being caused solely by the sudden re-action of the arterial columns of blood on the semilunar valves, its loudness will depend on the mobile and perfect state of these valves, and the extent and abruptness with which they are stretched by the recoil of the blood, at the moment of the ventricular diastole. It will therefore be most perfect when the heart acts regularly and slowly, giving time for the full gush of blood to carry the valves loose into their slight recesses in the walls of the artery, and for the as perfect reaction of the contents of the distended artery on their concave surfaces."* But if the second sound be produced by the columns of blood reacting against the semilunar valves, when the artery is distended, that must occur during the ventricular systole, for there are no columns of blood in the aorta and pulmonary artery during the ventricular diastole. The blood has already been propelled forward by the pulse in the aorta and pulmonary artery during the contraction of the ventricle; it is then only the blood that remains in the contracted arteries—that is in the arteries after the systole has taken place—that can fall back and recoil against the valves during the diastole. Dr. John Reid, whose statements are more explicit on this subject, says: "Synchronous with the first sound of the heart, we have the impulse of the heart against the chest, and the propulsion of the blood along the large arteries." "And during the second sound, which is synchronous with the diastole of the ventricle, we have the regurgitation of part of the blood in the large arteries upon the semilunar valves, throwing them inwards to the axes of the vessels." "And as the second sound appears to be produced by the shock of the blood upon the semilunar valves, its intensity must, in a great measure, depend upon the diastole of the ventricle drawing part of the blood back upon them; but perhaps more particularly upon the elasticity of the large arteries returning suddenly on their contents during the diastole of the ventricle, when the distending force of the ventricle has been withdrawn."* This observation shows that Dr. Reid, who performed numerous experiments on the action of the heart, considered that the reason usually assigned by physiologists for the cause of the second sound was insufficient to account for it, and therefore, he says, perhaps more particularly upon the elasticity of the large arteries returning suddenly on their contents. But we must observe, the ventricle during its diastole, cannot, by a sort of suction power, draw the blood back upon the semilunar valves with a force sufficient to produce an acute and sharp sound like the second sound of the heart. Besides, the dilatation of the ventricle always commences at the auriculo-ventricular foramen, and when the action of the heart is vigorous, the contraction of the auricles is synchronous with the diastole of the ventricle, and nothing like a suction power then exists in the ventricle to produce a reflux of the blood in the aorta against the semilunar valves. And as regards the elasticity of the arteries returning suddenly on their contents, that occurs during the systole of the ventricle when the blood is impelled forwards. During the ventricular diastole the arteries straighten themselves, and the aorta feels soft and compressible, and is in a state of rest, till its parietes are distended by the next ventricular contraction. And in the course of our experiments, no sound was produced in the aorta during the ventricular diastole.

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* Williams on "The Heart," p. 213.

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* "Cyclopædia of Anatomy and Physiology," London. Art. "Heart," by Dr. John Reid.

Physiologists seem to think that the contraction of the ventricle propels the blood along the arterial tube to its extremity, independently of the action of the semilunar valves; and that the blood in the distended arteries recoils against the valves during the diastole of the ventricle, and produces the second sound of the heart. But this is not the manner in which the heart acts. If the ventricle propelled the blood along the arteries, independent of the action of the aortic valves, it could not relax till the impulse which it imparted had reached the farthest extremities of the arteries, communicating the amount of the blood-wave to the veins, in which case the arteries would no longer be distended with blood; for if the ventricle relaxed before this was accomplished, then the blood would fall back, and recoil against the semilunar valves, and shut them, whilst it was in the act of being impelled along the arteries to their extremities, that is, during the arterial systole. But in transmitting the blood from the heart to and through the arterial system, the ventricle acts in concert with the aorta and its valves.[*]

And the aorta is not a rigid and unyielding tube, in which the fluid falls back against the valves when the propelling force has been withdrawn. But the aorta is distensible and elastic; and being distended by the blood-wave on contraction of the ventricle, instantly reacts or pulsates, forcing the blood against the valves as it is impelled onwards. And this occurs whilst the ventricle is contracted, and before the diastole has commenced. If there were no semilunar valves, the aorta being distensible and pulsative, would send the blood partially back into the ventricle at each pulsation; but the valves placed there prevent its return, and the impulse of the aorta which completes each successive systole of the ventricle exerts an influence in the movement of the blood throughout the arteries, the pulse, as we have stated, being felt at their extremities in an appreciable short interval after the aorta pulsates.

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[*] For the sake of illustration we may be permitted to allude to the action of the single forcing pump, which it somewhat resembles. The piston, which is contained in the working barrel at the base, has a range of two feet or upwards, and on being raised immediately descends to that extent, the valve opening, and the fluid passing readily into the upper compartment of the tube. But the instant the upward stroke is given the valve shuts by the fluid recoiling against it, and a distinct sound is produced; and as the piston is elevated to the limit of its range, viz., two feet, it exerts a force upon the whole of the fluid contained in the tube, expelling at the upper extremity an amount of fluid equal to what had entered at the base. And when the ventricle contracts it sends with an impulse the wave of blood into the aorta, distending its walls, and the aorta, reacting on the blood that has entered it, forces it against the valves, shutting them, and produces the termination of the first sound of the heart as the impetus is exerted on the wave, the impulse of the aorta being synchronous with the contraction of the ventricle, and preceding at a very short interval the pulse at the extremity of the arteries.

The fact, then, that the aorta is distensible and elastic secures its reaction on the blood, and shutting of the valves in connection with the ventricular systole.

When disease exists at the orifice of the aorta, interfering with the passage of the blood into the foramen, a bellows sound is produced, masking the commencement of the first sound of the heart; and if the aortic valves be incompetent a murmur is also produced as the aorta pulsates, affecting the termination of the first sound, the blood recoiling against the valves and partially regurgitating into the ventricle, so that the aortic regurgitant murmur occurs in connection with the ventricular systole, and not with its diastole, as is generally supposed. In proof of this we may observe, if the blood had been propelled by the ventricle along the arteries, independently of the action of the aortic valves, their incompetency could not affect the character of the pulse. But in all these cases the pulse is described as a "jerking pulse, the pulse of unfilled arteries, shooting like a ball beneath the finger," because the blood, instead of being sent along the arteries in a full stream, partly regurgitates through the aortic valves; just as the fluid in a pump is only partially expelled, when the valve in the working barrel is rendered incompetent. Physiologists are apt to think that this murmur, being regurgitant, must be of a diastolic character, and that it supplants the second sound of the heart; but it is quite the reverse. The blood only recoils against the valves from the impulse of the aorta that sends it onward, and the murmur is produced at the aortic incompetent valves as the wave advances. And in anæmia we have the bellows murmur well defined at the orifice of the aorta, and often more audible a little higher up as the aorta pulsates, but in all these cases the blood is passing along the aorta during the ventricular systole.

When disease commences in the aortic valves, and the individual is subjected to much physical exertion, causing the ventricle to contract and distend the aorta with energy, the reaction of the aorta on the blood-wave may be performed with an impetus which the valves cannot fully sustain, and regurgitation takes place partially into the ventricle. We perceive, then, how the first sound of the heart, produced in a state of health by the blood being propelled through the aortic foramen, and recoiling against the valves on the pulsation or reaction of the aorta, is converted in a state of disease into the aortic obstructive, and regurgitant murmur, which take place during the ventricular systole, as the blood is impelled along the arteries. We have even on several occasions, in the course of our experiments, when the action of the heart was weak, heard the first sound with a slight interval, as if it consisted of two parts. But this disappeared on the action of the heart increasing, and the ventricle contracting with greater vigour, the sound becoming continuous and distinct—the normal first sound of the heart. On one occasion when the action of the heart was not strong, the first sound was heard for a considerable time to consist of two parts, produced during the systole of the ventricle and pulsation of the aorta. Here there could be no reduplicate sound of the ventricle, and as regards the latter part of the sound, we could observe the blood recoil against the aortic valves when it was produced. The second sound was also occasionally heard, on contraction of the auricles, the three sounds constituting the triple sound of the heart. But when the ventricle contracted with more vigour the first became one continuous and distinct sound, and the second sound more audible, constituting the normal and healthy sounds of the heart.

When the ventricle contracts, the aorta pulsates, shutting the valves as the arterial systole takes place, immediately after which the ventricle relaxes as the auricles contract, the aorta becoming soft and compressible. But if during the diastole the arteries were distended with columns of blood that recoiled against the valves, it would act as an obstacle to the blood entering the aorta, and require an additional force to be exerted by the ventricle during its contraction to overcome it; and it would allow no period of rest to the parietes of the aorta, as they would be kept distended, both during the ventricular systole and diastole, which would speedily destroy their action.

There are three propositions, then, which we consider established by these experiments:—

1st. The first sound of the heart, viz., the dull and prolonged sound, must be associated with the contraction of the ventricle and pulsation of the aorta.

2nd. During the ventricular systole the semilunar valves are tightened and shut, by the blood recoiling against them on the impulse of the aorta.

3rd. The sound which is heard in the denuded heart over the semilunar valves or a little above them, is not the second sound of the heart, but the first sound attaining its intensity and termination.

The second sound has no connection with the aorta, but depends on the contraction of the auricles, and the force with which they pour their blood into the ventricles, and occurs quickly after the first sound terminates.

We have seen that closely following the first sound of the heart, a second sound is heard of a short, sharp, and acute character, and occurs during the contraction of the auricles and diastole of the ventricle, as was clearly ascertained by counting 1, 2, 3, 4, 5, 6, &c., whilst we listened to the second sound through the medium of the stethoscope; and several gentlemen carefully observed the contraction of the auricles, and each contraction of the auricles exactly coincided with the second sound as heard by us. Pressure on the aorta above the semilunar valves, at the moment of the occurrence of the second sound, did not in the least diminish its loudness and intensity, but it was affected by whatever impaired the action of the auricles. When they became weak, the second sound was scarcely audible, but when they contracted with energy, the second sound was clearly and distinctly recognised. It appeared to be produced at the auriculo-ventricular foramen, by the force with which the blood is propelled from the auricles into the ventricle. Sometimes it partook of a slight bruit, as if the blood passed over something rough on entering the ventricle. But when the auricles contracted with vigour, the second sound was sharp and acute, like the sound produced by the tongue striking the roof of the mouth. It appeared deeper seated, and at a greater distance than the first sound, because the auricles are here deeper seated than the arch of the aorta, and it did not follow the course and direction of the first sound. The first sound ascended along the aorta, but the second descended by the side of the base of the aorta, in the situation of the blood passing from the auricles into the ventricle. We tested this question with the greatest possible care, and distinctly ascertained that the second sound of the heart is produced by contraction of the auricles, as they pour their blood with force into the ventricles. After its occurrence a momentary silence ensued, and then the first sound recommenced.

We have also on several occasions, when the animal became weaker and the action of the heart less vigorous, heard the second sound double, as if the auricles did not contract exactly synchronous. July 21st.—Both sounds of the heart distinctly recognised, but the first is accompanied with a bruit that ascends along the aorta, and the second sound sometimes appears double, the contraction of the auricles being not exactly synchronous. July 23rd.—Both sounds distinctly heard, but the first is accompanied with a bruit, and the second sound more frequently double than formerly. August 3rd.—Action of the heart weaker, but both sounds can be fully recognised. Pulsation 28 per minute. The first sound attended with a louder bruit, and the second appears double, the auricles not contracting synchronously, and are slightly later in commencing. Three sounds are now heard—the first sound of the heart produced during contraction of the ventricle and pulsation of the aorta, and two short sharp sounds during contraction of the auricles; putting it beyond all doubt that in the normal and healthy action of the heart, the second sound depends on contraction of the auricles.

Dr. Hope concluded, as the result of his experiments on the sounds of the heart, that no sound is produced by contraction of the auricles. But this depended on the manner in which the action of the heart was maintained. We are informed that during these experiments, in which artificial respiration was employed, the auricles contracted only partially, or chiefly in their appendices. But to produce a distinct sound, the auricles require not merely to contract, but to contract with energy, in expelling the blood from their cavity. There are two circumstances which seem to affect the character and loudness of the second sound. When the action of the heart is weak, the auricles do not contract with a vigour sufficient to render the sound clear and distinct. And when the ventricle is already partially filled with blood passing into it from the distended auricles or regurgitating through the aortic valves, the auricle only partially contracts, and the second sound becomes obscure and indistinct. In these cases it is not heard constant and continuous. But when the action of the heart is vigorous, a sharp sound is produced by contraction of the auricles, distinctly recognised and closely following the first sound.

The principle which chiefly contributes to the production of sound in the contraction of the auricles is what obtains as a general law in the passage of fluids through narrow or contracted orifices. If the force by which the blood is propelled be weak, little or no sound is heard, but if the blood be propelled with energy, a clear and distinct sound is produced.

And Dr. Williams informs us, "that in some recent experiments with Mr. Clendinning, he found the auricles of an ass produce a very distinct sound when they contracted vigorously, and independently of the ventricles; this was afterwards heard by all who were present. The same phenomenon has also been observed in some experiments recently performed in America."* Drs. Pinnock and Moore heard a sound produced by contraction of the auricles.† And we have had ample evidence of this fact in the course of our experiments, during these three last summers, on the action and sounds of the heart in the American turtle.

Much has been said by physiologists respecting the second sound not being produced by contraction of the auricles, from the time at which the auricles are believed to contract. Harvey, Lancisi, Senac, and Haller considered that the auricles contract immediately before the contraction of the ventricles, and not immediately after the termination of the preceding contraction. Drs. Hope and Williams also maintained this doctrine as the result of their experiments; and Professor Turner,‡ arguing on these data, showed that as the second sound closely follows the first, it could not depend on contraction of the auricles, as their contraction immediately precedes that of the ventricles. Now, it is a fact, that when the action of the heart is slow and weak, as in cold-blooded animals, when it beats at the rate of 20 and 22 pulsations per minute, the ventricle after contraction immediately dilates, and blood enters it from the distended auricles, filling it out to a certain extent; and then the auricles contract and produce contraction of the ventricle, the one movement immediately preceding the other. The same thing occurs in warm-blooded animals after the thorax has been opened, when the action of the heart becomes slow and irregular, or its movements are interfered with, as by preventing the action of any of its valves. In these cases we have observed that after contraction, the ventricle immediately dilates to a certain extent, and blood passes into it from the distended auricles, and then the auricles contract and produce contraction of the ventricle. But we are by no means, from these data, to conclude that this is the manner in which the action of the heart is maintained, when it is quick and regular, beating, as in warm-blooded animals, at 60, 70, and 80 pulsations per minute. We have already shown* that as the action of the heart increases, the auricles contract sooner in point of time and of rhythm; then their contraction becomes synchronous with the diastole of the ventricle; so that they commence to contract immediately after the termination of the preceding ventricular contraction. And let a physiologist examine the action of the heart when it has been quickly denuded in a warm-blooded animal, and he will see that the auricles contract immediately after the termination of the preceding ventricular contraction, and that the action is maintained with surprising power, the one movement following the other in quick and regular succession. And this is exactly what we have seen in the course of our experiments on turtles during the highest temperature of the season.

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* Williams on "The Chest." † Wood's "Medicine." Philadelphia.

‡ Turner—"Med.-Chirurg. Trans." Edinburgh, 1829.

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* *British Medical Journal*, February, 1868.

In observing the action in a denuded heart, we distinctly perceive that the contraction of the auricles commence the cycle or beat, and the contraction of the ventricle and impulse of the aorta terminate the beat; the one beat succeeding the other in such quick and rapid succession that, when the action is vigorous, the contraction of the auricles is synchronous with the diastole of the ventricle. But in listening to the sounds of the heart through the medium of the stethoscope, or by the naked ear applied to the chest, we associate the dull and prolonged sound with the short and acute sound, considering the former the first sound of the heart and the latter the second sound, and that they belong to the same cycle or beat of the heart; but the reverse is the case. The short and acute sound is in reality the first sound of the heart, being produced by contraction of the auricles, which commence the cycle or beat of the heart, and the dull and prolonged sound is in reality the second sound of the heart, being produced by contraction of the ventricle and impulse of the aorta which terminate the beat; so that the short and acute sound does not belong to the same beat of the heart as the dull and prolonged sound which precedes it but is the commencement of a new beat, and must be associated with the ventricular contraction and impulse of the aorta that immediately succeed it; which enables us to see that the short and acute sound may appear to succeed the dull and prolonged sound, but is in reality the first sound of the heart.

We wish it to be distinctly understood that the statements we make respecting the sounds and rhythm of the heart refer exclusively to the action of the heart when it is quick and regular, and the contraction of the auricle immediately succeeds the contraction of the ventricle.

The first period of silence occurs between what is called the first and second sounds of the heart, and depends on the time at which the auricles commence to contract after the contraction of the ventricle and impulse of the aorta terminate. According to Müller it is one-fifth of a second; but Laennec thinks that the second sound begins immediately after the first ends. From careful and repeated observations, we are of opinion that an appreciable interval occurs between the first and second sound, that is, between the termination of the contraction of the ventricle and impulse of the aorta, and the commencement of the contraction of the auricles. But this interval is reduced to a minimum when the action becomes very rapid, the heart beating 100 and 120 times per minute.

The second period of silence occurs between what is termed the second and the first sound of the heart, that is, between the termination of the contraction of the auricles and contraction of the ventricles. It is longer than the first interval, because it extends through the period of the ventricular action, till the blood is sent with force into the aortic-foramen, and the semilunar valves shut.

From what has been stated we arrive at the following conclusions respecting the sounds of the heart :—

The first sound is produced by contraction of the ventricle and impulse of the aorta. It commences as the blood is propelled with force through the aortic foramen and attains its intensity in the aorta, as the blood is thrown back against the aortic valves and shuts them on the impulse being imparted to the wave.

This sound is heard most distinctly in the denuded heart over the semilunar valves—at the origin of the aorta or a little above it.

The second sound depends on the contraction of the auricles, and is produced as they propel their blood with force through the auriculo-ventricular foramen into the ventricle during its dilatation.

It appears to follow the first sound as an immediate sequence, as it takes place so quickly after its completion, but it is the commencement of a new beat, and synchronous with the dilatation of the ventricle, and of course precedes the ventricular systole.

And as the same principle obtains in warm-blooded animals, the first sound of the heart is produced during the ventricular systole, as the blood is propelled with force into the aorta and pulmonary artery, and attains its intensity, as the blood has entered their orifices and is thrown back against the semilunar valves and shuts them, by the reaction of the distended parietes imparting an impulse to the wave, the impulse of the aorta preceding the pulse at the wrist by an appreciable instant.

The second sound of the heart is produced by the force with which the blood is propelled by contraction of the auricle through each auriculo-ventricular foramen, into the ventricles during their diastole.

From what we have stated it will be seen that a bruit sometimes attends one or both sounds, when the action of the heart is weak, and disappears when the action is strong; so that one cause of inorganic murmurs is the want of energy in the contraction of the auricles or ventricles, and whatever restores this, removes the murmur.

From the manner in which the first sound is produced, we can perceive how a murmur connected with the contraction of the left or right ventricle is propagated along the aorta or pulmonary artery; a circumstance on which Dr. Hope laid great stress in determining to which side of the heart the murmur belonged.

We have thus given an account of the action and sounds of the heart, based on a series of carefully conducted experiments, where we had numerous and ample opportunities of investigating the subject, and we feel persuaded that what we have stated will ultimately be admitted to be the manner in which the sounds are produced. Dr. Hope, in the course of his experiments, limited the first sound to the action of the ventricle, and naturally thought that the sound which he heard in the aorta over the semilunar valves must be the second sound of the heart. But on this hypothesis it has been found impossible satisfactorily to account for the sounds of the heart; because, whatever view may be taken of the cause of the first sound, it occurs, as is admitted, when the ventricle contracts and the aorta pulsates, which makes the first sound of the heart synchronous with the pulsation of the aorta. But the aorta, when it pulsates, shuts the valves, and the sound which is then produced enters into the formation of the first sound, and constitutes its termination. Dr. Hope endeavoured to separate two things which are identical—the formation of the first sound, and the shutting of the aortic valves, which occur at one and the same moment, as the blood is impelled along the large arteries, and slightly precede the pulse at the wrist. So that what Dr. Hope considered the first and second sounds of the heart are simultaneous, and constitute one prolonged sound well brought out at its termination, recognised by physiologists as the first sound of the heart.

Whilst engaged in investigating the action of the denuded heart to ascertain the manner in which the diastole of the ventricle is produced, we perceived that when the ventricle contracted the aorta pulsated in connection with it, shutting the valves as the impulse of the blood-wave proceeded along the arch; and we concluded that the sound caused by the shutting of the sigmoid valves must occur sooner than is believed, and be synchronous with the ventricular systole. And on listening to the sounds through the medium of the stethoscope, we discovered that when the ventricle contracted it was one prolonged sound that was heard proceeding through the foramen into the aorta, where it terminates, being loudest at the aortic valves, or a little higher up. This, by repeated experiments, we distinctly ascertained to be what is termed the first sound of the heart.

Dr. Hope did not realize the true cause of the second sound of the heart, perhaps from the irregular manner in which the action was maintained. But we distinctly recognised the second sound, closely following the first, produced, as stated, when the auricles contracted and sent the blood with energy into the ventricle.

Much has been done by pathological research to establish the commonly-received views respecting the sounds of the heart; and yet pathologists still recur to the experiments that were first instituted to determine the cause of the sounds. And we may observe, if the first sound of the heart depended chiefly on the tightening of the muscular fibres of the ventricle, it ought to remain unaltered, under every circumstance, so long as the fibres are healthy and contract firmly. But the character of the sound is changed or modified in disease of the orifice of the aorta or affecting the valves.* And not only is this the case, but the reverse, the sound remains unaltered or little affected, even although the muscular fibres of the ventricle have, in places, entirely disappeared, being substituted by fatty globules; as fully ascertained by the microscope—the change, on the whole, "consisting more of fatty substitution rather than of fatty conversion." * And the first sound cannot properly be connected with the shutting of the auriculo-ventricular valves, as that takes place at the very commencement of the systole, whereas the first sound is admitted to be synchronous with the pulsation of the aorta, that occurs in connection with the blood-wave being propelled with force through the aortic foramen. And it is heard most distinctly in the denuded heart over the aortic valves; and seems to diminish as we approach the apex of the ventricle.†

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* Watson's "Practice of Physic," vol. ii. p. 234.

In diseased action of the aorta and its valves, a double murmur is produced, because a portion of blood regurgitates through the valves when the aorta pulsates. But in the healthy action, the first sound terminates when the sigmoid valves shut.

When the action of the heart is vigorous, the auricles contract with energy in propelling the blood into the ventricle. And if an obstacle exists to the free passage of the blood from the auricle into the respective ventricle, the auricles become dilated or hypertrophied.‡ And when the constriction of the mitral orifice has become extreme, the left auricle has been found enormously dilated and hypertrophied, and the second sound of the heart reduplicated §—a conclusive proof that the second sound depends on contraction of the auricles.

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* *British Medical Journal*, No. 570, p. 654.

† Carpenter's "Physiology," p. 419.

‡ *British Medical Journal*, No. 526, p. 92.

§ *Ibid.*, No. 573, p. 742.

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PREFACE TO THE SECOND EDITION.

In the second part of this Treatise, the author has more fully illustrated the principle on which the sounds of the heart are produced. And the question is not, how far the statements made by the author, may differ from the commonly received views of the cause of the sounds of the heart; but what does experimental research warrant us to consider the facts of the case? In every inquiry after truth, in medical science, views are only valuable, so far as they are established on facts.

London, *July*, 1874.

SECOND PART.

Physiologists have been accustomed to examine the action of the heart in cold-blooded animals, to determine the laws that regulate its movements. Dr. Hope performed a number of experiments on frogs and on turtles, to perceive the manner in which the auricles and ventricle contract and dilate. Professor Müller of Berlin, Oesterreicher, and Dr. John Reid prosecuted similar researches, and physiologists still continue to illustrate their views by experiments on the hearts of frogs and of turtles, as well as of warm-blooded animals, believing that the action is maintained on the same principle in both classes.

And convinced of the truth of this doctrine, we concluded that if the heart pulsate on the same principle in cold- as in warm-blooded animals, the sounds produced must be of the same character, and ought to be equally relied on. We therefore instituted a series of experiments on American turtles* during the highest temperature of the season, when the action of the heart is most vigorous, and the animal assumes the physiological condition of a warm-blooded animal; and we determined, with the greatest care, the manner in which the sounds of the heart are produced.

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* Alligator Terrapin. Chelydra Serpentina.

We have shown in the course of our experiments, recorded in the previous part of this treatise, that when the ventricle contracts and propels a wave of blood into the aorta, it distends its walls, which, being elastic, react with energy, and the blood recoils against the semilunar valves, and closes the orifice as the wave is impelled along the arch. The impulse of the aorta takes place in immediate connection with the contraction of the ventricle, and the first sound of the heart is heard most distinctly at the origin of the aorta, over the semilunar valves, and is produced by the force with which the blood is propelled through the aortic foramen and recoils against the valves closing them. Strictly speaking, the blood does not fall back against the valves as in a rigid and unyielding tube, but the aorta, on being distended by the blood-wave, starts up and contracts from its origin, the valves being closed by the blood recoiling against them as it is impelled onward. Hence the first sound of the heart is dull and prolonged, synchronous with the contraction of the ventricle and impulse of the aorta. And the second sound is produced by contraction of the auricles, and is heard most distinctly over the auriculo-ventricular foramen, as the blood is sent with force into the ventricle distending its walls. It closely follows the first sound, because during the vigorous action of the heart the auricles contract immediately after the aorta pulsates or reacts.

Having stated these facts, we proceed to explain more particularly the manner in which the action of the heart is performed and the sounds produced. The doctrines we maintain are, First, that the distended aorta reacts in immediate connection with the contraction of the ventricle, closing the sigmoid valves as its impulse is imparted to the wave. Second, the sound produced in closing these valves is the first sound of the heart attaining its termination; the sigmoid valves being closed at the commencement of the arterial systole, and not by a sort of back stroke, after the systole has taken place.

This statement we consider applicable to the action of the heart in all vertebrated animals.

Dr. Hope, in the experiments which he instituted on the denuded heart of an ass, found that a sound is produced by the blood recoiling against the sigmoid valves and closing them. But he believed this took place during the diastole of the ventricle, and was produced by a column of blood in the aorta and pulmonary artery falling back against the valves when the propelling force of the ventricle was withdrawn, and this would have been the case if the blood had been propelled along rigid and unyielding tubes; for the force exerted in propelling a portion of fluid into the tube imparts an impetus to the fluid which it contains, and an amount is discharged at the farther extremity equal to what has entered at the base. But on the impulse ceasing, on the ventricle commencing to dilate, its walls relaxing, the fluid would have fallen back against the valves and closed them. But the aorta is distensible and elastic, and according to

Hunter, the elasticity of an artery is greater at its origin—where the large vessels are connected with the heart, than at its distant extremity. Which somewhat affects the propulsion of the blood-wave. When the ventricle contracts and propels a wave of blood into the aorta, imparting an impetus to the blood which it contains, it distends its walls, which instantly react, and the blood recoils against the valves, and closes them as the wave is impelled onward. The force lost in the distension of its walls is now regained, and the ventricle is still contracted, when the aorta reacts or pulsates. And the view that the aortic valves are closed by the elasticity of the large arteries returning suddenly on their contents during the diastole of the ventricle, is opposed to the fact that the pulsation of the aorta takes place in immediate connection with the contraction of the ventricle, preceding the pulse at the wrist and extremities.

The question, 1st, which we have to decide is, when are the sigmoid valves closed ?

"Ces parois sont très élastiques; lorsqu'une ondée de sang est projetée dans l'aorte par la contraction du ventricule, elles cédent à la pression ainsi exercée comme le ferait un ressort, mais elles tendent ensuite à revenir sur elles-mêmes, et à chasser le sang qui les distendait."* And in all our experiments on the heart, whether in cold- or in warm-blooded animals—for we have frequently observed the action of the denuded heart in both—we have perceived that the moment the ventricle completes its contraction, the distended aorta reacts, forcing the blood against the valves, and closing them in impelling the wave onwards. And the blood could not be propelled in successive waves along a distensible and elastic tube like the aorta without the valves being immediately closed. When the ventricle contracts and sends the blood with force into the aorta, imparting an impetus to the blood which it contains, it distends its walls more particularly at its origin, where the greatest force and momentum is exerted. And if the aorta did not instantly react and impel onwards the blood which distends it, the pulse-wave would not be fully formed; or if the valves are not sufficiently closed, becoming incompetent, the same effect is produced. It is then at the commencement of the arterial systole, from the rapid reaction of the aorta, that the sigmoid valves are closed, and the blood impelled onwards.

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* "Zoologie," par Milne-Edwards, p. 70.

The sigmoid valves then placed at the origin of the aorta, aid and assist in the transmission of the blood-wave along the arterial tube. When the ventricle contracts, so much of the beat is completed, the blood having passed from the ventricle into the aorta. But the walls of the aorta are distended by the force with which the blood enters it, and must react before the wave is completed; and in reacting close the valves as a point of support against which the blood recoils in being urged onwards. The force of the contraction of the ventricle is the measure of its impulse, and the walls of the aorta react with a force equal or superior to that by which they are distended. And this takes place independent of the diastole of the ventricle, whilst it is still contracted. The impulse of the ventricle immediately succeeded by the impulse of the aorta, impels the blood-wave along the arteries to their termination. And the pulse-beat is felt before the second sound is heard. In applying our ears to the chest, we place our fingers on the pulse at the wrist, connecting its beat with the contraction of the ventricle, and the first sound of the heart.

It may appear remarkable that the ventricle should be contracted, when the blood recoils against the sigmoid valves, and closes the orifice. But the orifice is closed for the object of the blood being impelled forward—along the tube. And the ventricle, when it contracts, propels the blood-wave at once into the aorta, distending its walls, and does not go on contracting. It contracts to the extent of the impulse which it imparts at the moment, whether it expels the whole or the greater part of its contents, and its parietes are contracted when the aorta reacts and impels on the wave. But so rapidly is this accomplished, that when the ventricle propels the blood-wave into the aorta distending it, the aorta starts up or gives an impulse forward, and the reaction is completed. If the ventricle relaxed whilst the aorta reacted, it would be dilating during the propulsion of the wave onward, and before the systole was accomplished. For the pulse-wave is not fully formed before the aorta reacts. And the ventricle being contracted behind the valves must impart strength, as the blood reacts against them, stretching their segments.

We have thus the ventricular contraction, the aortic impulse closing the valves, and the pulse at the wrist; after which the second sound commences.

We therefore consider the view untenable which supposes that the sound produced by the blood recoiling against the aortic valves and closing them is the second sound of the heart, and coincident with the diastole of the ventricle. For the valves are closed as we have seen in immediate connection with the ventricular contraction by the aortic impulse which precedes the pulse at the wrist. And the sound heard on the closing of the valves is the first sound of the heart attaining its termination, which is a prolonged sound, produced by the force with which the blood is propelled through the aortic foramen, and the valves close as the aorta reacts or pulsates. When the diastole of the ventricle commences, its walls relax, and in unison with it the parietes of the aorta become soft and compressible, which is a state of rest; and at this moment there is no stretching or expansion of the sigmoid valves, nor falling back of the blood against them to produce a sound. And the experiment of the hooking back of a segment of the valves adduced so much in support of that view, produces exactly the same result, as is so frequently observed in the case of disease of the aortic valves when they are rendered incompetent. And regurgitant murmur is produced by a portion of blood being forced back into the ventricle during the impulse of the aorta. But this murmur does not represent the second sound of the heart, but the closing of the valves when the first sound terminates. And it does not occur at the time the ventricle usually dilates, but when the blood-wave is impelled along the arteries. And since the blood-wave is imperfectly formed, from the aortic impulse not being continued on it, the arteries are not sufficiently distended, and the pulse sudden without any prolonged swell of the artery. And the reason why the second sound is not heard is that the ventricle being partly filled by the regurgitation of the blood, the auricles do not contract with an energy in sending the blood through the ventricular foramen to produce a distinct sound.

And this condition of incompetency in the aortic valves is produced from the stress to which they are subjected when the distended aorta reacts with much force in propelling the blood-wave onward. If an obstacle exists to the transmission of the blood along the large arteries or through the arterial system, the ventricle exerts increased action to overcome it, in consequence of which it distends the aorta with greater force, and the walls of the aorta reacting with increased energy throw the blood against the valves with a force which they cannot sustain, particularly if diseased, and regurgitation of a portion of blood is produced into the ventricle. But this takes place as the blood-wave is sent along the arch, and not during the ventricular diastole.

In further proof of the fact that the aortic valves are closed in immediate connection with the ventricular systole, we may observe that in some animals, as the American turtle, there is an additional layer of circular fibres at the origin of the aorta, which fall back behind the valves as if to sustain them more when the blood in the aorta recoils, as the wave is impelled onward. Whilst in other animals, as the American sturgeon, a layer of muscular fibres is extended from the ventricle along the origin of the aorta, enabling it to contract with greater energy. And in adaptation to this increased power of the aorta, there are two rows or tiers of sigmoid valves placed at its origin, the one row immediately above the other. And a single tier of sigmoid valves—that is, of three valves placed a little higher in the aorta—opposite the termination of the external muscular fibres. The ventricle contracts and propels the blood into the aorta distending its walls, which instantly contract with energy, the blood recoiling against the double tier of valves and closing them as it is impelled onward, distending the aorta in front, which immediately reacts, forcing the blood-wave against the single tier of valves, and closing them as it is impelled along the tube. Here we have a double action in the aorta, closing two sets of valves in succession as it propels the blood-wave onward, which undoubtedly proves that the sigmoid valves are closed by the recoiling of the blood, as the distended aorta reacts and impels the wave along the arch. Affording one of the most striking evidences of design that we can contemplate, and illustrating a general law that obtains in vertebrated animals, that the blood, on being propelled from the ventricle into the aorta, recoils against the sigmoid valves and closes them as the elastic walls of the aorta react and impel the wave forward. And, in animals possessed of a single auricle and ventricle,* with one tier of aortic valves, and where no muscular fibres are extended along the origin of the aorta, the same effect is produced; the aorta, on being distended by the blood from the ventricle, instantly reacts, closing the valves as it urges on the wave.

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* Pisces.

In all these cases the valves are closed, as the blood in the distended aorta is impelled onward.

In warm-blooded animals the aorta at its origin is strongly elastic, and, on being distended, instantly reacts; and it is at the origin of the aorta that the force is chiefly exerted that propels the blood-wave along the arterial tubes. Just as in the working barrel at the base of the pump the upward stroke and movement of the piston imparts an impulse to the fluid in the tube, which at every stroke of the pump discharges an amount at the further extremity equal to what has entered at the base. The ventricle on contracting propels the blood-wave into and along the aorta, and the walls of the aorta are fully distended at their origin as the ventricle has completed its contraction, and react with such rapidity, closing the valves, that the impulse of the aorta may be said to be simultaneous with that of the ventricle; the forces thus exerted producing the blood-wave that passes along the arteries to form the pulse at their extremities. The impulse of the aorta slightly preceding the pulse at the wrist and extremities. We might even assume the major proposition in illustrating this subject. You cannot in a series of successive waves propel a fluid along a tube strongly elastic at the base without closing the valves at the orifice as the fluid enters. In propelling the wave into the tube, imparting an impetus to the fluid which it contains, the walls yield to the pressure and are distended. But the moment the wave has entered, the walls react with energy, closing the valves in giving an impulse to the fluid; and if a sound is produced in closing the valves, it must be when the walls react, urging the wave onward, as there is no recoiling of the fluid against the valves after the wave has been impelled along the tube and a quantum discharged at the extremity.

But if the force of the contraction of the ventricle distended the aorta and produced *per se* the pulse-wave at the extremities of the body, and the walls of the aorta reacted on their contents and closed the sigmoid valves in connection with the *diastole* of the ventricle, as is generally believed by physiologists, it is evident that the valves must be closed as a point of resistance to the blood when the elastic walls react; and in reacting they must displace the increased amount of blood which they contain, and produce a wave along the tube, which would render the second sound of the heart coincident with the blood passing along the arterial tube; whereas the pulse-wave is considered to be formed, and to have reached the wrist before the second sound occurs.* Besides, the ventricle, after its contractile impulse is exerted, cannot keep the walls of the aorta distended. They immediately react in virtue of the power which they possess as elastic tissue, their energy being superior to that of the force by which they were distended. When the ventricle then contracts and propels the blood-wave into the aorta, distending its walls, the valves are closed at the commencement of the reaction, the impulse beginning at the origin where the greatest force is exerted, and extending along the aorta, impelling the wave onward. And this enables us to see how the amount of blood which the ventricle propels at each successive beat exerts an influence on the pulse-wave throughout the arterial system; the ventricle propelling the blood along the aorta, and the aorta instantly reacting and continuing the impulse.

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* Müller's "Physiology," p. 176.

What is the condition of the sigmoid valves during the diastole of the ventricle? When the ventricle dilates, its parietes relaxing, the aorta takes on the same condition, becoming soft and compressible. And the valves having been closed by the recoiling of the blood-wave, remain shut till the ventricle contracts and opens them; the valves being opened by the contraction of the ventricle, and immediately closed by the reaction of the aorta. But observe, if the valves were closed during the diastole of the ventricle, by the blood recoiling against them from the elastic reaction of the great arteries near the heart, and the wave were not forced onward, it would keep the walls of the aorta distended both during the ventricular systole and diastole, which would soon destroy their elasticity, and produce the same result as disease of the walls of the aorta, when they are rendered incapable of effectually reacting on the wave and urging it forward.

The facts adduced enable us by strict induction to arrive at the following conclusions. 1st. When the ventricle propels the blood-wave into and along the aorta, it distends its walls more particularly at its origin, where the greatest force and momentum is exerted. 2nd. The distended aorta reacts in immediate connection with the ventricular contraction, closing the sigmoid valves as its impulse is imparted to the wave. 3rd. The closing of the sigmoid valves precedes the pulse-beat, of the facial artery—at the wrist—and extremities. 4th. The sound produced in closing the sigmoid valves is the first sound of the heart attaining its termination. 5th. The first sound of the heart is admitted by physicians to be synchronous with the contraction of the ventricle and reaction or pulsation of the aorta and large blood-vessels near the heart.*

II. We have based our views of the sounds of the heart, on the sounds that we heard in the denuded hearts of turtles, during the highest temperature of the season, when the action was maintained with great vigour at forty pulsations per minute. And we have stated, as admitted by physiologists, that the ventricle contracts and the aorta pulsates, on precisely the same principle in cold- as in warm-blooded animals —in the turtle as in the ass. And why should not the sounds of the heart be admitted to depend on the same principle in both? All the elements supposed to constitute or produce sound are present in the one case as in the other. The muscular walls of the ventricle contract with energy, and send the blood with force through the aortic foramen, and the blood recoils against the valves by the vigorous impulse of the aorta. And the sounds heard are often so clear and distinct that nothing can be more satisfactory, and bear the closest resemblance to the sounds of the heart in man and mammalia. To object to these sounds is to assume that vertebrated animals are not all constructed on the same type, and yet the only difference is, that the auricles pour the blood into one ventricle, which renders it not so highly arterialized as in the left side of the heart of mammalia, but more so than on the right, which is venous blood, and still the sound produced on contraction of the right ventricle is equally relied on, as that produced on contraction of the left.

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* Carpenter's "Physiology," p. 468. Müller's "Physiology," vol. i. p. 200. Hope on "The Heart," p. 56. Wood's "Medicine." Philadelphia.

Cruveilhier made observations on the action and sounds of the heart in the case of an infant only nine hours old, whose heart was exterior to the thorax, having passed through an opening in the upper part of the sternum;* and Dr. Carpenter adopted the data derived from the observations of Cruveilhier, as the basis for several of his conclusions respecting the action and the cause of the sounds. And physiologists have not objected to the principle of his argument, though they may differ from him respecting the manner in which the sounds are produced. But Billard informs us that he found, in examining the bodies of a great many infants who had died a few days after birth, the foramen ovale and other circulatory passages peculiar to the fœtus were generally closed about the eighth day; and that in nineteen infants who had lived only one day, the foramen ovale was completely open in fourteen.† Milne-Edwards also states, "Dans les premiers temps de la vie, tous les animaux à sang chaud, se rapprochent aussi plus ou moins des animaux à sang froid." ‡ So that we may conclude the infant on whose heart Cruveilhier made his observations partook much of the character of cold blooded.

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* Carpontor's "Physiology," p. 416.

† "Traité des Maladies des Enfants Nouveau-nés," p. 557.

‡ "Zoologie," par Milne-Edwards, p. 117.

We consider, then, that we have proceeded on sound physiological principles in the series of experiments that we instituted on the denuded heart of turtles during the highest temperature of the season, to determine the cause of the sounds of the heart. And we are decidedly of opinion that these animals afford a much better field for investigating the action of the heart, and arriving at a correct knowledge of the sounds than is obtained from the denuded heart of warm-blooded animals. In the warm-blooded, as in the dog and the ass, the operation of laying open the thorax and denuding the heart, produces a great shock on the system. And the fact that you require to maintain artificial respiration to continue your investigations for any length of time, interferes materially with the action of the heart, rendering it weak or irregular, and the sounds indistinctly heard. Hence the difficulty that experimenters have had in determining the cause of the sounds of the heart. But in turtles the effect of the operation on the action of the denuded heart appears but slight. If the temperature of the day be high, the heart continues to pulsate with great energy, and in a normal and regular manner after being exposed; and the animal will survive for several days, affording an ample field to observe the action and determine the cause of the sounds.

The auricles contract with vigour as in warm-blooded animals, distending the ventricle, and the ventricle propels the blood with force into the aorta, exerting its impulse on the wave as it is sent along the arch. But in listening to the sounds of the heart the action appears reversed, the dull and prolonged sound which is produced during contraction of the ventricle and reaction of the aorta is recognised as the first sound of the heart. And the short and acute sound which is produced during contraction of the auricles and dilatation of the ventricle, appears the second sound of the heart, closely following the first. But these facts could not have been determined without carefully observing the action of the denuded heart as we listened to the sounds. And it was distinctly ascertained in the course of these experiments, that the first sound of the heart represents the termination of the beat, and the second sound the commencement of a new or next beat, which seemed to follow the other as an immediate sequence, but depended on the force with which the auricles contracted and sent the blood through the auriculo-ventricular foramen into the ventricle. And the action was maintained without the occurrence of a pause—the auricles contracting with the commencement of the dilatation of the ventricle, and bringing it rapidly to the point of distension; the sounds occurring in the order we have mentioned, but a longer interval existing between the second and the first sound, because the first sound is produced when the ventricle propels the blood with force through the aortic foramen, and the aorta reacts. And this sound, as we have stated, is admitted by physicians to be synchronous with the contraction of the ventricle and pulsation of the aorta.

We conclude then that the sounds heard in the denuded heart of turtles, depend on the same principle as the sounds produced by the action of the heart in other vertebrated animals; and are equally to be relied on, as the sounds heard in the heart of the dog, the ass, the horse, and man.

From what has been stated, it appears physicians are proceeding on imperfect data respecting the action and sounds of the heart. That what is considered the cause of the second sound is not supported by fact; the sigmoid valves being closed in connection with the contraction of the ventricle, by the reaction of the aorta, as the blood-wave proceeds along the large arteries to produce the pulse at the wrist.

Much has been stated respecting the great advancement that physiological science has made in England and in Europe during the last quarter of a century. But it does not appear to consist in the increase of our knowledge on the sounds of the heart. The cardiograph has illustrated the action of the heart when it is slow, and the contraction of the auricles immediately precedes the contraction of the ventricle—a point to which Dr. Hope particularly directed attention in his researches on the action of the heart, and on which physicians still rely for an explanation of the cause of the sounds of the heart. But attention has not been so specially directed through the medium of the cardiograph as we could have wished, to the manner in which the action of the heart is increased and maintained when the contraction of the auricles becomes synchronous with the dilatation of the ventricle, and the movements are alternate without the occurrence of a *pause*. Though authors admit the fact.* Besides, the cardiograph throws no light on the cause of the sounds of the heart, but accepts the views of Rouanet, that the first sound of the heart is produced by the closing of the auriculo-ventricular valves on contraction of the ventricle, and the second sound on the closing of the sigmoid valves, by the blood in the aortic and pulmonary artery recoiling against them during the diastole of the ventricle,† an opinion generally entertained by French physiologists; whilst in England, as we have stated, physiologists believe that the sigmoid valves are chiefly closed by the elastic reaction of the large arteries near the heart, forcing the blood back against them during the diastole of the ventricle.

If the first sound of the heart were produced by the closing of the auriculo-ventricular valves, it is evident that it would be limited to the commencement of the contraction, when the fibres begin to tighten and the impulse to take place; and could not be persistent at the termination of the contraction and pulsation of the aorta. Dr. Carpenter observes* that no sound is produced by the closing of the auriculo-ventricular valves since they are restrained by the chordæ tendineæ, and do not fall back suddenly against each other, as is the case of the semi-lunar valves. And physicians give their united testimony to the fact that the first sound of the heart is synchronous with the contraction of the ventricle and pulsation of the aorta, thus connecting it with the termination of the beat. And when a murmur is produced from disease of the mitral valve, that murmur has its intensity at the apex of the heart, and persists only during a part of the ventricular systole.† Whereas the murmur arising from disease of the aortic orifice has its seat of greatest intensity over the third intercostal space, at the base of the heart, and is propa-

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* "En dehors même de tout désordre des mouvements du cœur, vous pouvez rencontrer dans son rhythme des variations très-notables. Ainsi que la circulation s'accelère, et vouz voyez la mesure se rapprocher davantage de celle à deux temps par la diminution et même la disparition du grand silence."—(p. 22.)

† "Lors de la diastole ventriculaire, la cavité, jusque-là effacée, reprend en peu d'instants ses dimensions premières, et le sang y affluerait à la fois par les orifices auriculo-ventriculaire et artériel, si ce dernier ne présentait par l'abaissement subit de ses valvules sigmoides (deuxième bruit) un obstacle au retour du sang de l'artère dans le ventricule." (p. 24.)—

"Leçons Cliniques sur les Maladies du Cœur."
Par J. Bucquoy. Paris.

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* Carpenter's "Physiology," p. 420.

† "Une lésion de l'orifice auriculo-ventriculaire gauche sera caracterisée de la manière suivante; un souffle systolique plus ou moins intense persistant pendant une partie de la durée du premier temps, avec son maximum d'intensité, à la pointe et se propagcant du côte de l'aisselle, indique une insuffisance mitrale.

"Passons aux lésions de l'orifice aortique, et comparons les données de l'auscultation. S'agit-il d'un rétrécissement, le bruit, qui est toujours assez rude, a son maximum d'intensité au niveau de cet orifice; c'est-à-dire, à la base; il est systolique et se propage

gated during the ventricular systole, most distinctly into the aorta and vessels of the neck, and sometimes even into a great part of the arterial system, which corresponds exactly to the description that Dr. Hope gives of the second sound of the heart. "The second sound is best heard over the semi-lunar valves, viz., on the sternum opposite to the inferior margin of the third rib, and thence for about two inches upwards along the diverging courses of the aorta and pulmonary artery respectively, the sound high up the aorta proceeding mainly from the aortic valves, and that high up the pulmonary artery being mainly from the pulmonic."* It is impossible to read this account of the second sound of the heart without perceiving that the sound occurred during the ventricular systole, since it is a law that sounds are propagated in the direction of the blood-current, which corroborates the fact that the first sound of the heart is produced at the origin of the aorta by the force with which the blood-wave is propelled into it, distending its walls, and they react, closing the valves in impelling on the wave.

When Dr. Hope listened to the sounds in the aorta, he believed that the blood was falling back against the valves, and closing them during the dilatation of the ventricle. But the blood was recoiling against the valves and closing them during the elastic reaction of

de la manière la plus évidente dans l'aorte et dans les vaisseaux du cou, quelquefois même dans une grande partie du système artériel." (p. 114.)—

"Leçons Cliniques sur les Maladies du Cœur."
Par J. Bucquoy. Paris, 1873.

* Hope on "The Heart," p. 64.

the aorta, in impelling onward the wave in connection with the contraction of the ventricle. We have seen that the aorta must react in connection with the ventricular systole as the blood is sent along the tube, for its reaction constitutes an important part in the impulse of the wave onward. . And during the ventricular diastole there is no falling back of the blood against the valves, and nothing to produce a sound, which annihilates the supposed cause of the second sound of the heart, and shows that the sound produced by the blood recoiling against the sigmoid valves takes place in connection with the ventricular systole. The ventricle is not relaxing when the aorta reacts. But the ventricle persists contracted * as the aorta closes the valves in completing the impulse on the wave. There can be no doubt that the aorta reacts in connection with the systole of the ventricle in forcing on the wave. Physicians admit the fact in explaining the principle on which it depends. The blood, after it has left the heart, says Dr. Watson, is urged onward by the healthy elasticity of the aorta.† But how could the elasticity of the aorta be exerted in urging onwards the blood without closing the aortic valves? If the valves are not closed, a part of the blood must regurgitate; and if the blood is impelled along the arteries,

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* "La systole ventriculaire persiste un certain temps après que la ligne d'ascension a atteint son summum, comme le demontre le plateau très-accusé du trace V. qui précède la ligne de descente." (p. 19.)—
"Leçons Cliniques sur les Maladies du Cœur."
Par J. Bucquoy. Paris.

† Watson's "Lectures on Practice of Physic," vol. ii. p. 228.

it must be during the ventricular systole, as we associate that act with the pulse-wave at the wrist and first sound of the heart. Besides, if the elasticity of the aorta be exerted in urging on the blood after it has left the heart, how could it be again exerted in causing the blood to recoil and close the valves during the diastole of the ventricle? The force once spent cannot be reproduced till the aorta is again distended by the next blood-wave.

From these facts we conclude that the blood does not recoil against the sigmoid valves during the dilatation of the ventricle, but in immediate connection with its contraction. And the sound produced by the closing of the valves is not the second sound of the heart, but the first attaining its termination. We must therefore look to another source to explain the cause of the second sound of the heart, and that is the contraction of the auricles.

III. The contraction of the auricles has been a subject of great difficulty to physicians, who have viewed the action of the denuded heart in living animals when it was slow, the sounds weak, and the contraction of the auricles immediately preceding that of the ventricle. They have concluded that this was the manner in which the action was always maintained, and that the second sound of the heart, heard on applying the ear to the chest, cannot be accounted for by contraction of the auricles, since it so closely follows the first or dull and prolonged sound. But physicians at the same time admit that when the action of the heart becomes vigorous, the pause disappears. The movements then become alternate. And from carefully conducted experiments we have distinctly ascertained that when the action of the heart is maintained with vigour, the contraction of the auricles commences immediately after the contraction of the ventricle and reaction of the aorta terminate, and the sound produced by contraction of the auricles so closely follows the first sound of the heart—the dull and prolonged sound—that it is viewed in connection with it. The first sound is produced at the termination of the beat, as the blood is propelled through the aortic foramen, and the distended aorta reacts or pulsates. And the second sound is produced at the commencement of a new or next beat, as the blood on contraction of the auricles is sent with force through the auriculo-ventricular foramen, being there most distinctly heard, so that the two sounds are viewed in close apposition. And this we consider the regular and normal action of the heart in a state of health.

But if the action of the heart becomes slower, the auricles are later in commencing to contract, and the parietes of the ventricle relaxing, a portion of blood passes into the ventricle, dilating its walls to a certain extent, and then the auricles contract and fully distend the ventricle, producing its contraction; the contraction of the auricles preceding the next ventricular systole. A slight pause may even take place after the partial dilatation of the ventricle, rendering the interval greater before the auricles contract and produce the contraction of the ventricle. And this is the phenomenon that physicians have so frequently observed in their experiments on the denuded heart. And it always indicates that the action is slow or irregular. We have distinctly observed in the course of our experiments that, on the heart being just denuded and its action vigorous, the auricles contracted at the commencement of the diastole of the ventricle rapidly distending its walls, and the second sound of the heart was clear and distinctly heard, closely following the first. But on the action of the heart becoming less vigorous, the auricles were later in commencing to contract. A portion of blood passing into the ventricle from the auricles and partially dilating its walls before the auricles contracted and distended the ventricle, and the sound produced on their contraction shortly preceded that of the ventricle. But the sound was now weak and merely distinguished, the auricles contracting with less energy.

We thus perceive that the auricles contract with vigour at the commencement of the diastole of the ventricle, producing the second sound of the heart; and when they are later in contracting, the ventricle being partially dilated, brings their contraction into nearer connection with the next ventricular systole preceding it. But the sound produced corresponds not in point of time or energy to the second sound as usually heard, the auricles contracting to a limited extent in exciting the contraction of the ventricle.

And the physiological fact that the second sound of the heart depends on contraction of the auricles is fully supported by pathological research. If an obstacle exists to the transmission of the blood from the left auricle into the ventricle, the auricle becomes hypertrophied from the increased action to which it is subjected—an indubitable proof that the auricles then contract with energy; and when the orifice of the mitral valve becomes constricted, a bruit is produced on contraction of the auricle during the diastole of the ventricle. Not what is strictly termed a presystolic bruit, but a bruit coincident with the ventricular diastole,* which bruit we consider represents the second sound of the heart.

The non-attention to the principle on which the action of the auricles is maintained, or not having listened to the sounds of the denuded heart when the movements of the auricles and ventricles are alternate, has been the cause of inaccurate views on this subject.

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* "La théorie nous avons montré qu'un bruit de souffle au second temps ou diastolique perçu à la pointe appartient au rétrécissement mitral et le caractérise." —(p. 37.)

"Pour vous en convaincre, lisez le remarquable mémoire publié dans les *Archives générales de médicine* (1853, 1854), par mon éminent collègue M. Herard, sous ce titre, *Des signes stethoscopiques du rétrécissement de l'orifice auriculo-ventriculaire du cœur*, *et spécialement du bruit de souffle au second temps;* vous y trouverez la démonstration clinique du fait que j'avance."—(p. 38.)

"Le souffle, répondant à la systole auriculaire, dans quelques cas, *peut-être perçu nettement pendant le second temps*" (Herard).—(p. 114.)

"Le premier temps commence avec la systole ventriculaire, et à la même durée; le second répond à la diastole ventriculaire." (p. 19.)—

"Leçons Cliniques sur les Maladies du Cœur."
Par J. Bucquoy. Paris.

Instead of viewing the auricular systole as taking place after the partial dilatation of the ventricle, we must identify it with the commencement of the diastole of the ventricle, which brings it into immediate succession with the first sound of the heart. It represents the state of the circulation when the action of the heart is maintained with vigour and not when it is slow or irregular.

According to the rapidity with which the circulation is maintained, so is the contraction of the auricles. If the action of the heart be vigorous, the auricles contract immediately after the reaction of the aorta terminates, producing the second sound of the heart. And the whole of the blood that enters the ventricle is projected by contraction of the auricles. If the circulation still increase and be maintained at a high rate of speed, immediately succeeding the arterial systole, the auricles shoot down the blood with an impulse into the ventricle, rapidly distending it, and instantly relax as the ventricle contracts. It may therefore be stated as a general law, that the rapidity with which the auricles contract after the reaction of the aorta, indicates the energy in the action of the heart.

From this statement of facts, the result of experiments carefully conducted and repeated, we conclude that the first sound of the heart is produced at the origin of the aorta, by the force with which the ventricle propels the blood into it, distending its walls, and the aorta reacts, closing the valves in impelling onward the wave. It is the termination of the beat; and the second sound so closely following the first, is the commencement of a new beat, and produced by contraction of the auricles as they send the blood with force through the auriculo-ventricular foramen into the relaxing parietes of the ventricle and distend it.

The manner in which the sounds of the heart are produced may be thus stated, premising that the observations which apply to one ventricle or one side of the heart are equally applicable to the other.

First sound produced by	Ventricular contraction and Aortic reaction.
Second sound, produced by	Auricular contraction, as the ventricle dilates.

We have no theories to support, *hypotheses non fingo*. We have stated facts and doctrines based on these facts by strict and rigid induction.

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